THE MEDICAL JOURNAL OF AUSTRALIA

VOL. II.—38TH YEAR. SYDNEY, SATURDAY, SEPTEMBER 8, 1951. No. 10.

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INTRAHEPATIC BILIARY TRACT OBSTRUCTION OF UNKNOWN ORIGIN: THE SO-CALLED CHOLANGIOLITIC HEPATITIS.

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THE whole problem of liver disease and its sequelæ received much attention during and following the 1939-1945 war. We are particularly indebted to Lucké (1944) for his clinical studies of 125 American servicemen who died of infective hepatitis. Virchow's conception of a catarrhal inflammation of the papilla of Vater, an ascending infection, giving rise to the old term "catarrhal jaundice" was shown to be too narrow an impression by many individual observers, notably Eppinger and Klemperer. The Swedish school with Iverson and Roholm (1939), and the British Postgraduate Medical School with McMichael, Dible and Sherlock (1943) and many others added confirmation to the hepatitis philosophy, using the modern aspiration liver biopsy technique. Wood *et alii* (1948) have demonstrated similar results in Australian subjects.

The clinical picture and biochemical findings in infective hepatitis were accurately defined, and it was generally considered that most cases ended in full recovery, a small percentage ended fatally, and a smaller group smouldered on to the development of hepatic cirrhosis. It is this fascinating small group that progresses as "chronic hepatitis" that has occasioned such careful study during the last few years.

Watson and Hoffbauer (1946) described a rare sequela to infective hepatitis to which they gave the name "cholangiolitic hepatitis". The term cholangioles includes the finer biliary radicles outside the lobule, especially the ampullæ of the bile capillaries and the primary bile canaliculi in the portal spaces. They reported eight cases which presumably began as ordinary hepatitis (not actually proven), but in which jaundice was prolonged for many months and in one case for forty-three years. The clinical, biochemical and pathological picture that developed differed greatly from that normally found in chronic hepatitis. After the initial febrile episode jaundice was prolonged for months, but without much general disturbance. Despite deep jaundice their patients were relatively well. The jaundice was of the obstructive type, with dark urine and pale stools. Both liver and spleen were generally enlarged, sometimes grossly, but ascites was absent. Liver function tests showed remarkable normality. As time passed these patients presented more and more the signs of obstruction, but at operation no such obstruction was found. The condition of these patients must then represent an intrahepatic obstruction, which could be due theoretically to periportal cellular accumulations, bile thrombi, pressure from actual increase in the size of the liver cords, or leakage of bile through damaged cholangioles with return of bile to the blood-stream. The pathology in these cases makes the first three hypotheses unlikely, and the authors consider the cause to be a severe functional derangement of the cholangioles. Their account of the pathology reveals generally a surprisingly normal histological picture despite severe jaundice. The lobular pattern was preserved, and the hepatic cells appeared normal. Bile-staining of the hepatic cells and bile thrombi within and between the cells were seen. There was periportal infiltration, but it was not pronounced. In a few

cases many of the liver cells were multinucleated. There was no necrosis, no loss of reticulum, no pronounced leucocytic infiltration, fibrosis or bile-duct proliferation. Cases 5, 7 and 8 of their series were of particular interest, as the patients developed cirrhosis, the final histological picture being indistinguishable from that of portal or Laennec's cirrhosis. The patient in Case 8 was known to have pronounced cirrhosis eight years before she died.

In Karsner's well-known paper (Karsner, 1943) on the different forms of hepatic cirrhosis, he quotes Roessle on cholangiolitic cirrhosis:

> Primary injury occurs in the finest biliary passages within the lobules with consequent injury to the parenchyma cells. Biliary obstruction is primarily intralobular, but there may be proliferation of ducts in the portal spaces and also formation of papillæ within them. The perilobular connective tissue is slightly if at all increased in amount and is loosely arranged. Fibrosis within the lobules may be conspicuous. Infiltration by lymphocytes, plasma cells and large mononuclears is accompanied by a considerable exudation of polymorphs, more conspicuous within the lobule than within the portal space. The plugs of inspissated bile occur in the intralobulary biliary passages rather than in the ducts of the portal space. Thus in this form of lesion the changes as a whole are more marked *in the lobule* than in the portal space, whereas in the cholangitic form the reverse is the rule.

These cases of "cholangiolitic hepatitis" are to be distinguished from other causes of obstructive jaundice, whether intrahepatic or extrahepatic, and the infective or "cholangitic hepatitis". The latter is described excellently by MacMahon (1931), who points out that the infection is most pronounced within the terminal bile ducts, that there is a lack of uniform general distribution and that inflammatory changes and necrosis occur early.

Xanthomatous biliary cirrhosis was originally believed by Thannhauser to be a primary disturbance of cholesterol metabolism, but recently MacMahon and Thannhauser (1949) have reversed their opinion and now believe this syndrome to be a primary disease of the liver with extremely high cholesterol readings. Dauphinee and Sinclair (1949) describe the same thing. MacMahon and Thannhauser's description of the histopathology in these cases is strikingly similar to that in cases of cholangiolitic biliary cirrhosis in Watson and Hoffbauer's series. However, the authors state that the nature of the reaction may be distinguished from changes seen in obstructive and cholangiolitic biliary cirrhosis, although they do not state what the differences actually were.

These uncommon forms of hepatic disease have revived interest in Hanot's original descriptions; to quote Karsner again:

> It seems probable that what Hanot actually saw was a disease which is now recognized as either intrahepatic cholangitic or cholangiolitic cirrhosis. Thus these forms of cirrhosis may be looked upon as the modern counterpart of Hanot's hypertrophic cirrhosis.

The following case would appear to fit into this group of intrahepatic biliary tract obstruction of unknown origin, the so-called cholangiolitic hepatitis syndrome.

Report of a Case.

CASE I.—A male patient, while serving in the Australian Military Forces in February, 1944, at the age of twenty-three years, as an infanteer, became a little nauseated and off his food. It was noticed that his conjunctiva had a yellow tinge and that his liver was just palpable, and he was admitted to hospital with a fever. Malignant tertian malaria parasites were found in his blood, and he was given a course of antimalarial therapy. His gastric discomfort disappeared and he felt reasonably well. His weight was 10 stone. His urine, however, remained dark, and his stools became pale and the jaundice increased in depth. After sixty days he was still deeply jaundiced, but felt well, taking food without nausea or discomfort. There was no history of gastro-intestinal disease prior to this illness. He had not lost any weight. The abdomen was soft, without tenderness, and no masses were palpable. The liver and spleen could not be felt. He was considered to be suffering from infective hepatitis. Laboratory findings at this time included the following. The icteric index varied from 40 to 90 units. The plasma bilirubin content was 10 to 12 milligrammes *per centum*. The Van den Bergh reaction was strongly positive in both direct and indirect reaction. Urobilinogen was present in the urine to excess. The total red blood cell count was 4,400,000 per cubic millimetre. The hæmoglobin value was 12·8 grammes *per centum*. The hæmatocrit value was 58%. The total white blood cell count was 7000 per cubic millimetre, made up of neutrophile cells 32%, eosinophile cells 1%, lymphocytes 62%, basophile cells 1%, and monocytes 4%. Red cell fragility tests (repeated several times) revealed that hæmolysis began at 0·45% strength of salt solution and was complete at 0·3% strength. The reticulocyte proportion was less than 1%. The bleeding time was two minutes. The coagulation time was three minutes. The prothrombin time was 80% of normal. Blood Wassermann, Eagle and Kahn tests all yielded negative results. The Casoni test result was negative. Several examinations of stools for cysts, ova *et cetera* failed to reveal anything significant. The finding from a hippuric acid liver function test was 2·2 grammes' excretion in four hours (73% of normal). The total serum protein content was 7·2 grammes *per centum*. The urine constantly contained both bile pigments and urobilinogen.

By August, 1944, the patient was still deeply jaundiced and the liver was now palpable two fingers' breadth below the costal margin. It was now felt that the jaundice might be obstructive and the patient was referred to a surgeon. In September, 1944, laparotomy was performed. Some glands along the common bile duct were enlarged and one was removed for biopsy. The common bile duct was of normal size and the gall-bladder was not distended. The spleen was of normal size, but the liver was greatly enlarged, of regular contour and of firm consistence. A biopsy was taken. There was no evidence of any extrahepatic obstruction.

Histological examination of the lymph gland showed chronic inflammatory changes and the biopsy of the liver was reported upon at that time as showing "moderately advanced hepatitis". When sections were recut and reviewed at a later date quite a different pathological picture was seen.

By March, 1945, the patient was still in hospital and still jaundiced, and the results of laboratory investigations were similar to those previously described. He was "boarded out" of the army, medically unfit, with a guarded prognosis.

During the succeeding two years he was reasonably well, with a mild degree of jaundice. He suffered from occasional abdominal pain situated in the epigastrium and right hypochondrium. In May, 1946, the gall-bladder was twice submitted to X-ray examination by the Graham method, and in both instances the dye did not fill the gall-bladder. The radiologist reported the gall-bladder as being pathological.

These attacks continued intermittently, but the patient continued to work hard on an orchard until November, 1948, when he was admitted to the Repatriation General Hospital, Concord, with severe pain in the epigastrium and right hypochondrium. He was found to be moderately jaundiced, his weight was 10 stone, he was slightly tender in the epigastrium and right hypochondrium, the liver was palpable one finger's breadth below the costal margin and the spleen was not palpable. The urine contained bile. Findings of laboratory investigations were as follows. The total red blood cell count was 5,000,000 per cubic millimetre. The hæmoglobin value was 14·5 grammes *per centum*. The reticulocyte proportion was less than 1%. The prothrombin time was 78% of normal. The leucocytes numbered 9150 per cubic millimetre, with a normal distribution. Red cell fragility was normal. Blood Wassermann and Kline tests yielded negative results. Casoni and hydatid complement fixation test results were negative. The serum protein content was 7·8 grammes *per centum*. The plasma bilirubin content was 4·4 milligrammes *per centum*. The thymol turbidity test result was two units.

A plain X-ray picture of the abdomen was normal, and as on the two previous occasions, a Graham's test failed to show any gall-bladder shadow on X-ray examination. It was felt that the clinical story was suggestive of a stone in the common bile duct with gall-bladder disease, and the patient was examined in consultation by the surgeon who had previously operated upon him.

A laparotomy was performed in January, 1949. The liver was found to be dark green in colour. There were dense abdominal adhesions, and the gall-bladder was intrahepatic. The common duct was opened, but no calculi were seen and no extrahepatic obstruction could be demonstrated. The common bile duct was dilated and a portion of liver removed for biopsy. The gall-bladder was removed and it was found to contain numerous small facetted gall-stones. The biopsy findings will be discussed later.

After operation the patient had a most stormy convalescence. A large loculated hæmorrhage occurred around the operation site, followed by bile peritonitis. The jaundice became intense and the patient wasted. After energetic and persistent treatment he fully recovered and returned to the medical ward—still mildly jaundiced.

Laboratory findings in March, 1949, were as follows. The plasma bilirubin content was 3·5 milligrammes *per centum*. The total serum protein content was 6·7 grammes *per centum*, made up of serum albumin 4·6 grammes *per centum* and serum globulin 2·1 grammes *per centum*. The thymol turbidity test result was two units. The serum alkaline phosphatase content was 5·2 King-Armstrong units. Examination of the stools for fat (with the patient having a 50-gramme fat diet) revealed a total fat content in dried stool of 30%, made up of split fat 14% and unsplit fat 16%. The glucose tolerance test yielded the following values for blood sugar content, in milligrammes *per centum*: fasting, 80 (repeated, 70); after half an hour, 86 (repeated, 84); after one hour, 89 (repeated, 94); after one and a half hours, 105 (repeated, 90); after two hours, 100 (repeated, 79). The urea clearance test yielded a value of 92% of normal.

The patient was weak, but now quite comfortable, without pain, and he gained weight rapidly. After a prolonged convalescence he was finally discharged from hospital in May, 1949, after a stay lasting six months. He was still mildly jaundiced and his weight was nine stone two pounds.

At review in July, 1949, he had been playing "doubles" at tennis without distress, his weight was nine stone eleven pounds, he had no pain or indigestion, and his stools were normal in colour. He was mildly icteric, the liver could be felt one finger's breadth below the costal margin, and the spleen was not palpable.

He was again reviewed in January, 1950. He had been working for the past five months as a storeman without loss of time. His weight was nine stone six pounds, and he had remained well. He was still mildly jaundiced, and the clinical findings were essentially the same.

His last review was in August, 1951. His weight was ten stone, and he had continued working in the same capacity without loss of time. He had remained well and able to do most things without effort. He was still as jaundiced as before, and the clinical findings were unaltered. The biochemistry findings were as follows. The plasma bilirubin content was 3·7 milligrammes *per centum*. The serum alkaline phosphatase content was 6·1 King-Armstrong units. The total serum protein content was 7·8 grammes *per centum*, made up of serum albumin 5·4 grammes *per centum* and serum globulin 2·4 grammes *per centum*; the albumin-globulin ratio was 2·3:1·0. The thymol turbidity test yielded a value of 11 units. The serum cholesterol content was 112 milligrammes *per centum*.

Biopsy Reports.

Examination of the first specimen obtained at biopsy in September, 1944, shows a liver architecture which is relatively normal. There is pronounced bilirubin staining of the liver cells, especially in the centre of the lobules. In the periportal connective tissue a moderate number of lymphocytes and, to a less extent, plasma cells can be observed. In one area there is a group of multinucleated liver cells suggesting regeneration secondary to injury. The bile ducts appear normal. There is no evidence of hepatic cell necrosis, fatty change or fibrosis (Figures I, II and III).

Examination of the second specimen obtained at biopsy in January, 1949, shows remarkably similar changes. The bilirubin staining of liver cells is even more pronounced, but the periportal infiltration is much less. The bile ducts are normal and there is no increase in connective tissue (Figure IV). The histopathological picture is in keeping with the picture of cholangiolitic hepatitis as described by Watson and Hoffbauer.

A Warning Case.

The inadvisability of accepting this diagnosis in relation to a patient aged over forty years is well shown in the following clinical history.

CASE II.—A male patient, aged forty-five years, after serving within Australia with the Royal Australian Air Force during the war, suffered from dyspepsia and was granted a pension on the basis of a duodenal ulcer in May, 1946. He remained reasonably well until January, 1948, when his appetite became poor and he developed lassitude and tired easily. During the next month he lost seven pounds in weight; his stools became pale and his urine dark. He had a dull pain over the liver on admission to hospital. Examination revealed slight jaundice and an enlarged liver, two fingers' breadth below the costal margin. The spleen was not felt. The temperature was normal.

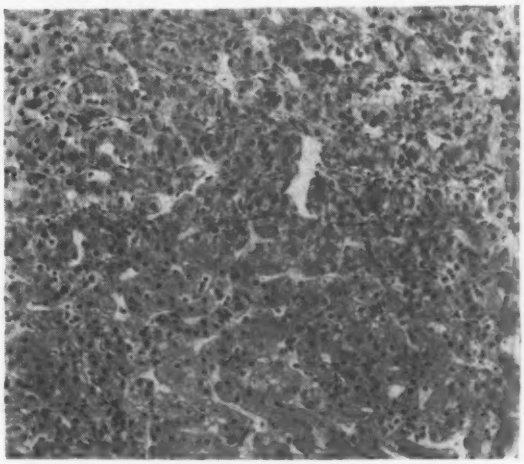

FIGURE I.

Case I, biopsy I. Liver lobule with central hepatic vein showing round-cell infiltration. There is no centrilobular necrosis. Bilirubin staining of the hepatic cells is well seen in the original section. (Hæmatoxylin and eosin stain, × 100.)

Laboratory tests in April, 1948, yielded the following results. The total red blood cell count was 5,290,000 per cubic millimetre. The hæmoglobin value was 15·3 grammes *per centum*. The total leucocyte count was 5600 per cubic millimetre, made up neutrophile cells 68%, eosinophile cells 1%, lymphocytes 29%, monocytes 2%. The hæmatocrit value was 49%. Plasma bilirubin values were 1·4, 1·7, 2·1, and 1·3 milligrammes *per centum*. The alkaline phosphatase content was 32·6 King-Armstrong units. The thymol turbidity test yielded a value of one unit. Urobilinogen was present in the urine. The hippuric acid test yielded a normal value. X-ray examination after barium meal revealed no abnormality.

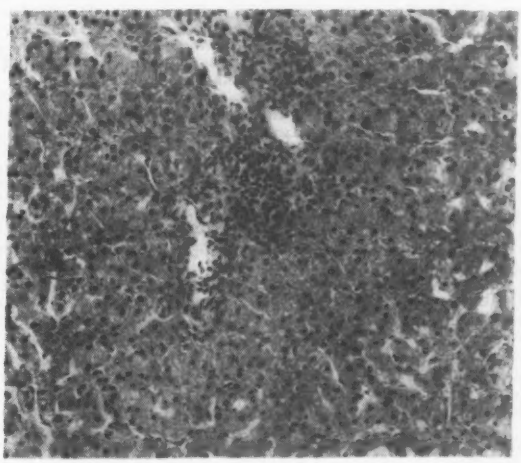

FIGURE II.

Case I, biopsy I. Showing the periportal lympholytic infiltration and bilirubin staining of liver cells. (Hæmatoxylin and eosin stain, × 100.)

The patient was discharged from hospital in May, 1948, with doubtful mild icterus and feeling reasonably well. His next admission was in March, 1949. During the ten months' interval he remained well except for intermittent attacks of intense pruritus, malaise, anorexia, pale stools and dark urine, lasting from a few days to one to two weeks. He also had dull pain in the epigastrium and right hypochondrium. Two X-ray studies of the gall-bladder were carried out by the Graham technique during the three weeks prior to his readmission, and in both cases the gall-bladder failed to fill.

On admission to hospital in March, 1949, he was slightly icteric, the liver was now grossly enlarged six finger's breadth below the costal margin and appeared to have an irregular surface. The spleen was felt on inspiration and was firm.

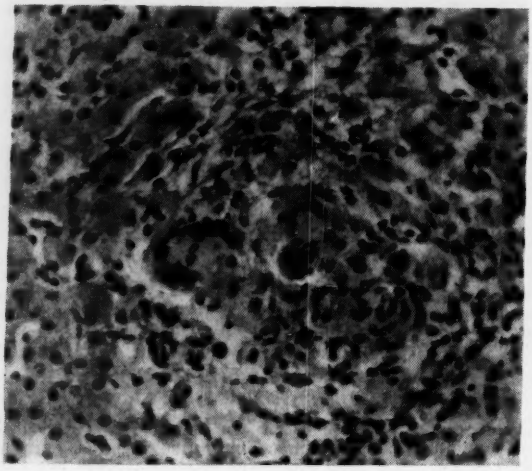

FIGURE III.
Case I, biopsy I. Showing the multinucleated liver cells. (Hæmatoxylin and eosin stain, × 400.)

Laboratory examination findings were as follows. The plasma bilirubin content was 0·5 milligramme *per centum*. Urobilinogen was present in the urine as a trace. The total serum protein content was 6·8 grammes *per centum*, made up of albumin 4·5 grammes *per centum* and globulin 2·3 grammes *per centum*. The thymol turbidity test yielded a value of one unit. Blood Wassermann and Kline tests yielded negative results, as did the hydatid complement fixation, amœbic complement fixation, and Casoni tests. Examination of stools (several) failed to reveal ova or cysts. Examination of the fat content of stools with the patient receiving a 50-gramme fat diet revealed 30·6% of total fat, 24·5% of split fat, and 6·1% of unsplit fat. In the erythrocyte fragility test hæmolysis began at 0·48% and was complete at 0·32%. The proportion of reticulocytes was 0·5%. In a Graham's test, performed for the third time, the gall-bladder failed to fill. The following blood sugar values, in milligrammes *per centum*, were obtained from a glucose tolerance test: fasting, 75; after half an hour, 102; after one hour, 129; after one and a half hours, 87; after two hours, 79.

The patient was discharged from hospital after three weeks, feeling reasonably well, and continued to eat a diet containing 120 grammes of protein. He remained fairly well except for further intermittent attacks of similar nature to those already described. The pruritus was particularly troublesome. In August all the symptoms recurred with jaundice.

He was admitted to hospital for the third time in August, 1949. The liver felt even larger and projected as an obvious tumour. It felt irregular. In view of the recurring attacks it was thought that there was probably obstruction to the common bile duct, and the surgeon who operated twice in Case I was asked to examine this patient.

Laparotomy was performed on August 16, 1949. The liver was found to be much enlarged and its surface finely granular. The gall-bladder was distended, and the common bile duct was dilated to about three-quarters of an inch in diameter. The gall-bladder was opened and found to contain yellow bile, some small facetted stones and biliary mud. The common bile duct was explored; when it was opened, bile spurted out under greatly increased pressure. The common bile duct contained some thick biliary material, but a probe passed down easily into the duodenum. Sounds also passed easily. No extrahepatic obstruction could be found. In view of the stormy convalescence in Case I and the doubtful benefit of the cholecystectomy in that case, it was decided to perform cholecyst-duodenostomy, and a tube was also sutured into the common bile duct and brought out via a stab wound in the right flank. After the operation there was copious bile drainage from this tube for two weeks, as much as 160 ounces of bile being recovered in one day. Despite this, the stools were coloured a dark brown. The patient came to present a problem in fluid and electro-

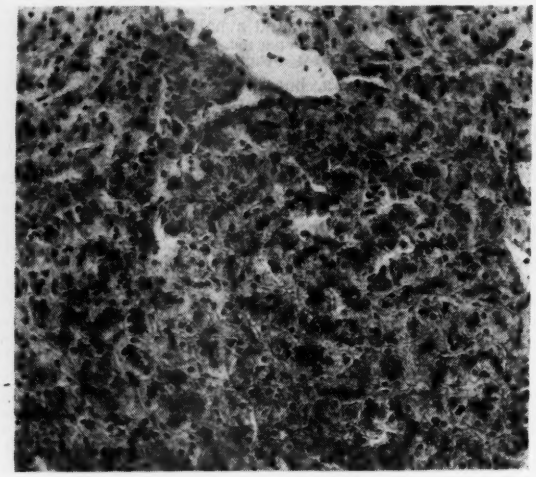

FIGURE IV.
Case I, biopsy II. Showing a similar pathological picture to the first biopsy. No cellular necrosis but more bilirubin staining of liver cells. (Hæmatoxylin and eosin stain, × 100.)

lyte balance and was cholæmic for several days. The tube was taken out on the fourteenth day, and to our surprise no bile leaked out through the wound, and he made a rapid recovery. Examination of the section of liver removed at operation showed hepatic cirrhosis which conformed to the cholangiolitic type described by Watson and Hoffbauer.

The patient was reviewed at regular monthly intervals and remained well until March, 1951. There had been no recurrence of his jaundice. The liver was now barely palpable, and the spleen could just be felt on inspiration. There were no spider nævi, liver palms or ascites. When he was last examined, in February, 1951, the laboratory test findings were as follows. The plasma bilirubin content was 0·4 milligramme *per centum*. The serum cholesterol content was 164 milligrammes *per centum*. The alkaline phosphatase content was 6·2 King-Armstrong units. The thymol turbidity test yielded a value of two units. The total plasma protein content was 6·8 grammes *per centum*.

However, in May he suffered a severe hæmatemesis and melæna, and on examination a tumour was felt to the right of the mid-line above the umbilicus. Laparotomy disclosed a mass surrounding the pancreas, and examination of a biopsy specimen revealed an anaplastic carcinoma derived from a glandular structure.

Biopsy Report.

Examination of the piece of liver tissue removed at operation in August, 1949, shows an increase of perilobular connective tissue. The lobules are variable in size and completely surrounded by the young connective tissue, and the cirrhosis tends to be of the multilobular type. The connective tissue contains fibroplastic proliferation and is infiltrated with chronic inflammatory cells. The small bile ducts have undergone proliferation. Plugs of bile pigment are present

in the bile capillaries. The changes may represent the end stage of cholangiolitic hepatitis (see Figure V).

Examination of a biopsy specimen of liver tissue in June, 1951, revealed relatively normal hepatic architecture with portal scarring and some fine fibrosis.

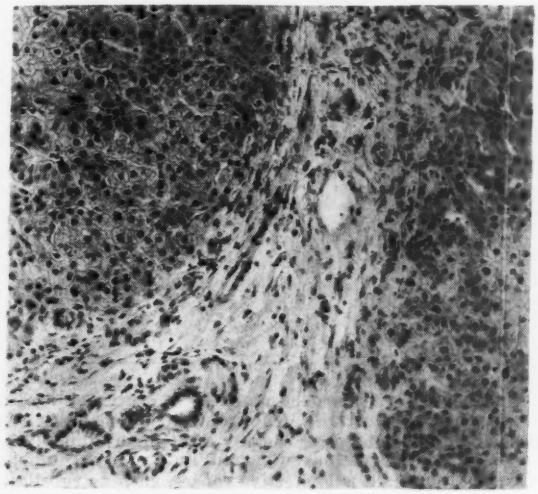

FIGURE V.

Case II. Showing a cirrhosis of multilobular type indistinguishable from portal cirrhosis. Plugs of inspissated bile are present in quantity. (Hæmatoxylin and eosin stain, × 100.)

Discussion.

The two cases described represented a type of obstructive jaundice, but at operation no extrahepatic obstruction could be demonstrated.

If biliary obstruction is allowed to continue for long, hepatic cirrhosis will follow. Cameron (1935) has shown experimentally that biliary obstruction results in an interference with the regenerative capacity of the liver cells, and MacMahon and Mallory (1929) have reported a careful study of 30 cases of uncomplicated obstructive cirrhosis. We have been impressed by the operative results in our two cases. In Case I the operation of cholecystectomy appears to have had no influence on the obstructive lesion, whereas in Case II the operation of cholecyst-duodenostomy seems to have played a major role in the remarkable clinical and pathological recovery that resulted for almost two years. We have been impressed by the Swedish reports on their results of surgical procedures in chronic hepatitis with obstructive signs. Bergenfeldt (1947) describes seven cases of prolonged jaundice due to apparently severe hepatitis, in which perfusion of the biliary ducts with normal saline resulted in sudden and permanent improvement. He suggests that in such cases an accumulation of tough mucus and bile causes a relative obstruction in the lower part of the common bile duct. When the biliary pressure rises sufficiently, bile seeps down into the intestine and pressure is relieved. Hublin (1948) also describes four cases of severe hepatitis successfully treated by irrigation of the bile ducts.

Guillet in Paris, cited by Brattsom (1949), states that: "... atonia of the sphincter of Oddi is a usual concomitant of hepatitis but that occasionally hypertonia is encountered in prolonged icterus in hepatitis as shown by roentgen manometric measurements with increase of pressure in the common bile duct of more than 16-20 cm. water. The significance of the hypertonia may be in the relative stasis in the biliary passages which sustains and aggravates the hepatitis." Brattsom (1949) reports on cases of prolonged hepatitis in which surgical drainage was performed (external choleductus drainage in all but one in which a cholecyst-duodenostomy was performed). Nine of the patients were completely well five to ten years later.

It would seem, therefore, that in these cases of hepatitis with continuing jaundice, which the French call *"ictère médicale prolongé"*, some form of surgical drainage with perfusion of the bile ducts might be considered.

Summary.

Two cases are presented of a type of intrahepatic obstructive jaundice of unknown ætiology which appeared to correspond to the so-called cholangiolitic hepatitis of Watson and Hoffbauer. After two years' observation Case I would still appear to fit this diagnosis; but Case II has revealed itself as carcinoma, the site of origin of which is still obscure. The diagnosis of cholangiolitic hepatitis should not be readily accepted, therefore, in relation to a patient aged over forty years. The patients concerned were subjected to surgery. The relevant literature concerning surgery in prolonged hepatitis is discussed and a suggestion made that surgery might be considered in cases of chronic hepatitis in which there are obstructive features.

Acknowledgements.

We should like to thank the Commissioner for Repatriation for permission to publish this paper and for placing facilities at our disposal. We should also like to thank the following for their helpful advice and criticism: Dr. K. W. Starr, who performed the surgical operations in the two cases; Dr. T. J. Constance, pathologist of the Repatriation General Hospital, Concord; Dr. A. W. Morrow, of the Royal Prince Alfred Hospital; Dr. Ian Wood, of the Walter and Eliza Hall Institute of Medical Research, and Dr. Sheila Sherlock, of the British Postgraduate Medical School, London. We are indebted for the photomicrographs to Mr. Woodward-Smith, of the Department of Medical Artistry, Department of Medicine, University of Sydney.

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MORTALITY FROM CONGENITAL MALFORMATIONS IN AUSTRALIA.

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THIS paper examines the deaths from congenital malformations in relation to the fall of mortality in Australia over the years 1908 to 1945, and tests the possibility that any substantial proportion of these deaths are due to epidemic causes affecting the *fœtus in utero*.

Sources of Data.

The deaths from congenital malformations were included under a single title of the "International List of Causes of Death", and were not further analysed in the Australian statistical bulletin, *Demography*, until the year 1940, after which the classification was carried out according to the fifth revision of the International List. Thus, here, for the period 1941 to 1945, the deaths from congenital malformations are broken down into eight named subdivisions with a ninth subdivision devoted to "other congenital malformations", but for the years 1908 to 1940 they are included in a single title. In the search for epidemicity, the deaths from the New South Wales "Statistical Register" were examined, as it might be that epidemics in a single State would be swamped by inclusion in the Australian figures. Dr. N. K. Dougan has kindly put some further clinical material at my disposal on the incidence of cleft palate and hare-lip.

Congenital Malformations as a Cause of Death.

In Table I are given the numbers of deaths from congenital malformations by age in Australia. Evidently in most of the cases so diagnosed the subject dies in the first year of life. It is shown later in Table III that almost half the total deaths occur in the first week of life. Few subjects are certified as dying from congenital causes after the end of the first year of life. Some congenital defects are not included in this class at all, a notable example being mongolism, cases of which are included in the class of nervous diseases. There is a reluctance among those responsible for the design of the International List and among the coders in the statistical offices to assign deaths occurring later in life to this class. Thus congenital defects of the aorta giving rise to subacute bacterial endocarditis would be assigned to the infective cause or disease rather than to the congenital lesion.

In Table II the effect of the congenital malformations on the infant mortality rates is noted. To ensure uniformity with the tables of my other papers on mortality, the rates are given as deaths *per annum* per million births; but the rates are readily transformed into rates per thousand births. The death rates from congenital malformations have remained relatively constant over the years considered, 1908 to 1945. They were lower in the two earliest periods, 1908 to 1910 and 1911 to 1920, but this was probably due to under-certification, the deaths being certified as due to congenital debility or other cause outside this class.

The masculinity of the rates has been determined by dividing 100 times the male rate by the female rate. The masculinity of the rates has ranged from 114 to 129. The male rate is about one-quarter higher than the female rate. Of course, no adequate explanation of this phenomenon is available at present.

In Table II, in the two last columns, it is noted that the congenital malformations are playing a relatively larger part in the infant mortality in recent years than formerly. This is obviously due to the fall in the death rates from most of the other causes. Comparisons between different series or countries are best made by the use of the number of deaths per 1,000,000 or 1000 live births (or per total live births and stillbirths as is sometimes possible). Confusion must arise if the mortality rates from congenital malformations are compared by the use of the ratios of the deaths from these causes to deaths from all causes at these ages, for the infant mortalities in many countries are in a state of rapid transition to lower rates. For this reason confusion has resulted in Chapter III of the Ministry of Health Report Number 94 (1949).

TABLE I.

Deaths from Congenital Malformations in Australia.

Period.	Sex.	Number of Deaths at Ages (Years).			
		0	1 to 4.	5 and Over.	Total.
1908 to 1910 ..	M.	541	44	9	594
1911 to 1920 ..	M.	2696	229	85	3010
1921 to 1930 ..	M.	3239	209	175	3623
1931 to 1940 ..	M.	3030	265	341	3636
1941 to 1945 ..	M.	1754	186	97	2037
1908 to 1910 ..	F.	427	26	7	460
1911 to 1920 ..	F.	2023	202	77	2302
1921 to 1930 ..	F.	2397	207	174	2778
1931 to 1940 ..	F.	2226	254	313	2793
1941 to 1945 ..	F.	1464	192	124	1780

In Table III are given the numbers of deaths by age for two periods in Australia. The point to be made is that most of these deaths occur shortly after birth; thus, if the congenital malformations occurred in any great number of cases from maternal infective causes, such as rubella or influenza, the epidemic waves might be expected to occur after the style of those demonstrated for the births of the deaf (Lancaster, 1950). Since death may not follow immediately after birth, an epidemic wave, due to an epidemic of infective disease affecting mothers, might be expected to begin sharply and then to tail off slowly or more slowly than the curve of the births of the deaf.

TABLE II.

Mortality from Congenital Malformations in Australia.

Period.	Mortality Rates[1] per Million per Annum, in First Year of Life.		Masculinity of Mortality Rates for the First Year of Life.	Proportions of All Infantile Deaths that were Certified as Due to Congenital Malformations.	
	Males.	Females.		Males.	Females.
1908 to 1910 ..	3077	2605	118	3·76	3·82
1911 to 1920 ..	4014	3171	127	5·38	5·28
1921 to 1930 ..	4713	3681	128	7·73	7·57
1931 to 1940 ..	5069	3915	129	11·37	11·13
1941 to 1945 ..	4661	4088	114	12·01	13·22

[1] These divided by 1000 give the mortality rates in the same units as is customary for infant mortality rates.

The Epidemicity of the Deaths from Congenital Malformations.

Since the discovery that rubella causes some congenital effects, such as congenital heart disease, other infective causes have been examined, and Professor L. Dods suggested to me that the official statistics and hospital admissions should be examined for evidence of epidemicity. To this end the monthly incidence of deaths in Australia from congenital malformations has been examined for the years 1908 to 1945. Any variations can be explained as sampling fluctuations. In particular, the influenza epidemic of 1919 and the rubella epidemics of 1938 to 1941 have not made any appreciable epidemic waves. However, as it appears possible that the epidemics in different States might not have occurred at the same time, the deaths in New South Wales of infants aged under one year from congenital malformations by month of occurrence have been compared for the years 1940 to 1947, by the use of the published tables from the "Statistical Register", the official publication of the Government Statistician, New South Wales. No evidence

for epidemicity of the individual types of congenital malformations can be found, with the exception of congenital abnormality of the heart. It appears that 1941 may be regarded as an epidemic year for congenital diseases of the heart in New South Wales, for the observed deaths exceed the expected in the months near the centre of the year. The observed numbers of deaths are given by month with the expected numbers in parentheses as follows: eight (11·1), nine (8·4), eight (10·2), eight (8·5), 18 (13·3), 20 (13·7), 13 (12·9), 24 (16·0), 11 (12·7), six (10·7), seven (11·0), six ((9·5). The expected number has been determined by computing the total number of deaths in each month from the same cause for the years 1940 to 1947 in New South Wales, and then dividing the result by a factor

TABLE III.

Mortality from Congenital Malformations in Australia.

Period.	Sex.	Number of Deaths at Ages.				
		Under One Week.	In Rest of First Month.	In Rest of First Year.	At All Other Ages.	All Ages.
1911 to 1920 ..	M.	1310	543	843	314	3010
1911 to 1920 ..	F.	988	394	646	279	2302
1941 to 1945 ..	M.	829	319	606	283	2037
1941 to 1945 ..	F.	699	253	512	316	1780

so that the total of the expected is the same as that of the observed total for 1941 in New South Wales. If the 1941 figures of monthly deaths are compared with those of the other years 1940 and 1942 to 1947 combined by a conventional statistical test, the χ^2 test for homogeneity, it is found that even these differences are not significant, for χ^2 is 17·39 for 11 degrees of freedom; so that the increased incidences in certain of the months of 1941, although suggestive of an epidemic wave, are not conclusive. It certainly follows that in New South Wales even in 1941 not a very large proportion of the deaths under one year of age from congenital malformations of the heart could have been due to rubella or other infective cause. I have examined the Queensland deaths from congenital malformations from this point of view, and there are no variations in the number of deaths in the years 1940 to 1947 suggestive of epidemic occurrences. It appears that the following conclusion is justified for the congenital malformations sufficiently serious to cause death within a few months of birth. There is no evidence of epidemic occurrence in the Australian statistics in the years 1908 to 1945. In particular, the influenza epidemic of 1919 did not give rise to an epidemic of congenital malformations serious enough to cause death within a few months of birth. Nor is it likely that any disease occurring epidemically in Australia can be responsible for the production of any great proportion of the deaths from this class of diseases through its action on the *fœtus in utero*. In other words, it would appear that the vast majority of the lethal congenital abnormalities occur independently of one another, and are due to causes peculiar to the individual pregnancy—that is, to hereditary causes, to maternal causes and to unspecified chance factors such as mutations and accidents.

Incidence of Hare-lip or Cleft Palate or Both in Melbourne.

Dr. N. K. Dougan has obtained and has kindly allowed me to use some statistics bearing on the problem of epidemicity of congenital disease. He has found that for the period July, 1934, to June, 1949, there were 596 patients with cleft palate, or hare-lip, or both in combination, operated upon at the Children's Hospital, Melbourne. Of these patients 374 were males and 222 females. The month of birth of each patient was obtained or estimated from the clinical notes when it was not specifically stated. I have been unable to detect any evidence of epidemic waves, nor is there any special seasonal occurrence. These findings are in accord with the conclusions reached from a study of the official statistics, already mentioned.

The Type of Congenital Malformation.

In Table IV are given the deaths from the various types of congenital abnormality in Australia at all ages for the years 1941 to 1945. It is evident that congenital malformations of the heart account for some half of the deaths, and that congenital hydrocephalus accounts for about an eighth. It is possible that the congenital hydrocephalus deaths are understated, since if such subjects die from meningitis, as they are prone to do, the death may be

TABLE IV.

The Deaths Classified by Type of Malformation, Australia, 1941 to 1945.

Type of Abnormality.	Number of Deaths in Australia, 1941 to 1945, by Sex.			Expected Number of Males Computed on Basis of Births from 1941 to 1945.
	Males.	Females.	Total.	
Congenital hydrocephalus	249	220	469	240
Spina bifida and meningocele	133	148	281	144
Congenital malformation of the heart	1016	914	1930	989
Monstrosities	63	75	138	71
Congenital pyloric stenosis	(91)[1]	40	131	67
Cleft palate, hare-lip ..	32	23	55	28
Imperforate anus ..	(37)[1]	15	52	27
Cystic disease of the kidney	15	10	25	13
Other congenital malformations	401	335	736	377
Total	2037	1780	3817	1956

[1] These two malformations show a significant degree of masculinity.

referred to the infective condition. A similar warning applies to the *spina bifida* and meningocele subdivision. In practically every subdivision of the deaths in Table IV there is an excess of male births over the number which would be expected from the sex of the total number of infants born, on the hypothesis that the malformations occur with equal likelihood in a child of either sex. The masculinity is significantly high in the case of congenital pyloric stenosis and imperforate anus. Moreover, the deaths in both these subdivisions and in the cleft-palate-hare-lip subdivision are only a small proportion of the total number of cases and represent the deaths due to operative mortality, or to neglect, or to inability to receive the treatment in time for it to be effective, and it may be that there is a differential mortality rate operating there. On the other hand, the masculinity is low, although not significantly so, in the cases of *spina bifida* and meningocele and of monstrosities.

As no effective measures can be used against the class of deaths due to congenital malformations in the present state of knowledge, they form a lower limit to the possibilities of lowering the infantile death rate. But this limit —about four deaths per 1000 births—is much lower than the present infant mortality rate.

Summary.

The deaths in Australia from congenital malformations have been analysed. The death rate in the first year of life from these malformations has remained relatively constant over the years of the survey, 1908 to 1945. As the infant mortality from all causes has dropped considerably in the same period, the mortality from congenital malformations is assuming greater relative importance as a cause of death in infancy in Australia. There is a pronounced masculinity of the deaths from this class of diseases, particularly the deaths from congenital pyloric stenosis and imperforate

anus. No conclusive evidence of relation to epidemic disease is apparent in the Australian statistics, although there is a suggestive rise for congenital abnormalities of the heart in New South Wales coincident in time with the epidemic of births of the deaf in the middle months of 1941. It is therefore concluded that epidemic disease plays a relatively small part in the production of these abnormalities.

Acknowledgements.

This paper is published with the permission of Dr. A. J. Metcalfe, Director-General of Health, Canberra.

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THE SYDNEY REPRINT OF JENNER'S "INQUIRY".

By EDWARD FORD,

School of Public Health and Tropical Medicine, University of Sydney.

IN 1884 a reprint of the second edition of Edward Jenner's "An Inquiry into the Causes and Effects of the Variolæ Vaccinæ" (1800) was published by the Government Printer, Sydney, for official distribution (Figure I). Except for a prefatory notice in the book, no published reference to the reasons for this Australian appearance of the famous quarto is known, though these are of interest to both student and collector. It is now also generally forgotten that this reprinting of the "Inquiry" was associated with the long and unsuccessful campaign for compulsory vaccination that was waged by New South Wales medical practitioners for many years. For these reasons it is proposed to recall briefly the circumstances leading up to the publication of the reprint.

When the practice of vaccination was introduced by the publication of the "Inquiry" in 1798, the first settlement at Port Jackson had already been established for ten years. The rapid and widespread adoption of Jenner's method was reflected in the new colony, for by the arrangement of Governor King "vaccine matter" was received from England in 1804, and successful vaccination was first performed by the Principal Surgeon to the Colony, Thomas Jamison, and by Surgeon John Harris and Assistant-Surgeon Savage. A scheme for voluntary vaccination was instituted, but from the earliest days the medical practitioners of the Colony expressed their dissatisfaction with the poor public response. This formed the subject of the first medical paper to be published in Australia—a contribution to the *Sydney Gazette* of October 14, 1804, entitled "General Observations on the Small-pox", by Principal Surgeon Jamison. Medical writing, on the same theme, continued to appear till the end of the century.

By 1860 all the Australian colonies, with the exception of New South Wales and Queensland, had enacted legislation for compulsory vaccination. In this they followed Britain and many other countries. Numerous attempts were made to secure the introduction of similar legislation in New South Wales. This was strongly recommended by a succession of medical advisers to the Government, who appear to have been firmly supported by the local medical profession. Small-pox had been absent from the Colony for many years, and voluntary vaccination had dwindled to a level which gave grave fears for the consequences should the disease again be introduced. "There is . . . an opposing moral force against which all professional energy and all ordinary reasoning operate without effect", wrote Dr. Francis Campbell, the medical adviser to the Government, in his report for 1867.

Under the conditions which prevailed at the time, especially in regard to health services, medical care and communications, and with a rapidly increasing and scattered population, there existed serious grounds for the fears that were constantly expressed by medical men. In view of the existing legislative precedents, based on the best medical opinion, and the strong recommendations of their advisers, the remissness of successive New South Wales governments in not enacting compulsive legislation would be difficult to understand were the vagaries of State politics not comprehended. Tidswell (1898) ascribed their resistance to three causes: popular fear of the possible coincidental inoculation of syphilis or other diseases; the failure of vaccination to check the spread of small-pox on certain occasions, though unvaccinated persons had been numerous; and undue reliance upon maritime quarantine, in the absence of any Federal quarantine organization.

In 1869 a suggestion was received from the Secretary of State for the Colonies that vaccination should be made compulsory in New South Wales. The following year the Governor recommended to Parliament that legislation should be enacted for that purpose. A Bill, which was largely similar to the Victorian *Act to Make Compulsory the Practice of Vaccination* of 1854 was introduced. In the same year the Bill, with amendments, was reported to the House by a committee to which it had been referred, but was then allowed to lapse. This was the nearest approach to enactment ever made by such a measure in New South Wales.

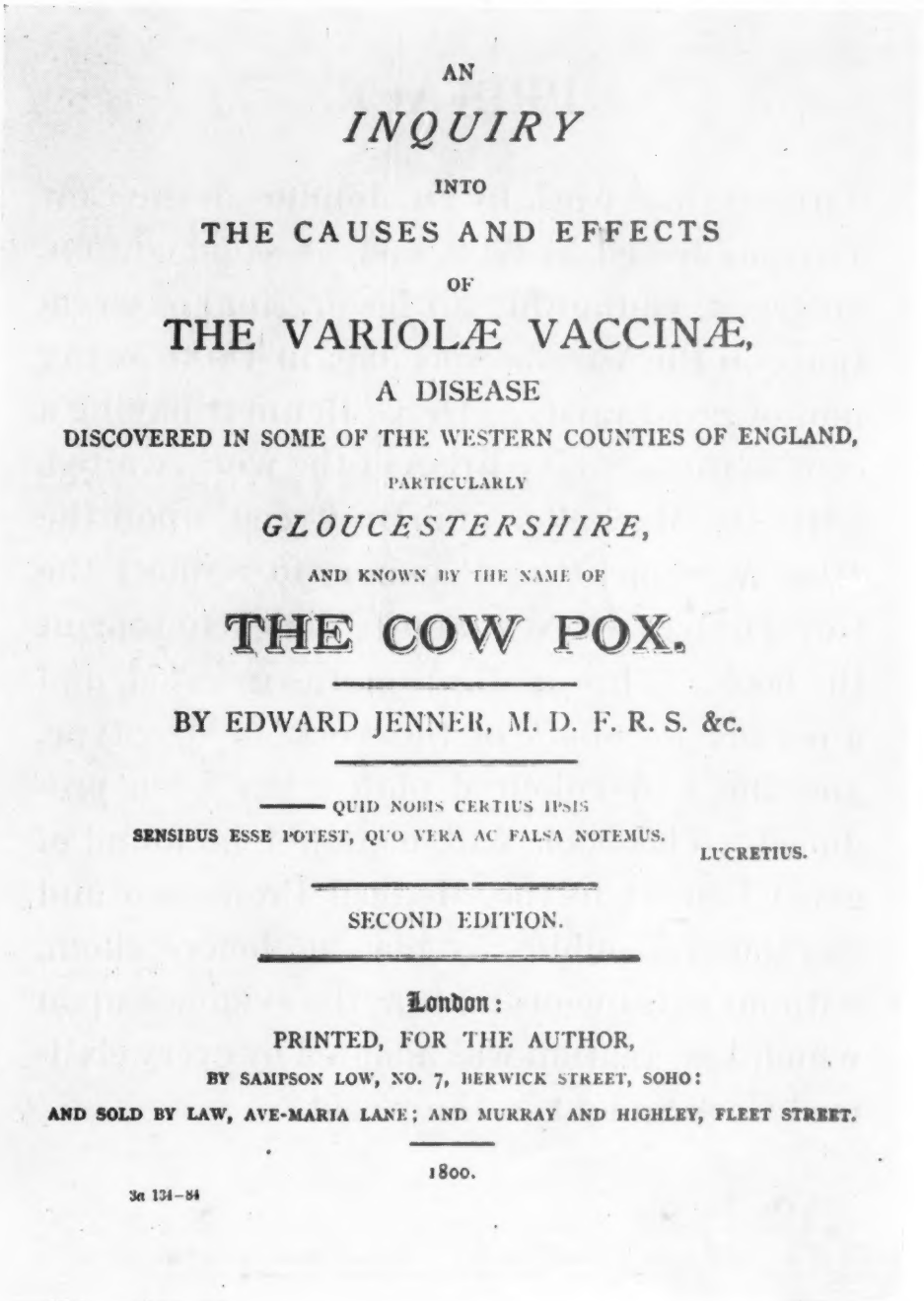
For many years no incentive to legislative action was provided by the occurrence of small-pox in the Colony. Outbreaks occurred in Victoria in 1857, 1868-1869 and 1872, but except for a single case at Newcastle in 1874 New South Wales remained free of the disease from 1830 to 1877. In that year 12 cases, with four or five deaths, occurred in Sydney, and a further fruitless attempt was made to secure compulsory vaccination. There followed, in 1881-1882, the most serious epidemic of small-pox ever reported in Australia. This also occurred in Sydney and accounted for 154 cases, with 40 deaths. In 1883 a single case was recorded, and in 1884-1885 a further epidemic of 64 cases, with four deaths. Cumpston (1914) considered that the latter was probably a continuation of the 1881-1882 epidemic.

During this period of small-pox incidence, agitation for vaccination legislation was intensified in New South Wales. In 1881 the Cabinet met as a Committee of Inquiry, and evidence upon the value of vaccination was taken from many medical practitioners. No action resulted, however, and the dictum that "pestilence is a hasty law-maker" in this instance failed to be fulfilled. The publication of the Jenner reprint was associated with this phase of the vaccination campaign, in which the influential medical men named in the preface of the work (Figure II)—Dr. G. Bennett, Dr. C. K. Mackellar and Dr. C. M. Creed—played an important part.

George Bennett (1804-1893), from whose original copy the reprint was made, was a physician of high academic and scientific attainments. He was a member of the Faculty of Medicine of the University of Sydney and a consulting physician to Saint Vincent's Hospital. Throughout his busy medical life he was an important worker and collector in the field of natural history, and was the author of many medical and scientific contributions. Cleland, in an interesting account of Bennett's life and work in this journal (1950), regarded him as perhaps the most distinguished of our medical naturalists.

At the time of the publication Dr. (afterwards Sir) Charles Kinnaird Mackellar (1844-1926) was first president of the recently established Board of Health, and medical adviser to the Government. Like previous medical advisers, he strongly recommended compulsory vaccination. In 1882, as president of the Board of Health, he issued a pamphlet on the preparation of calf lymph, which was then replacing arm to arm vaccination (Figure III). The greater part of the booklet, however, was devoted to a Bombay *Act for the Compulsory Vaccination of Children,*

which was presumably inserted in the hope that it would serve as the pattern for a local statute. Mackellar resigned from the Board of Health in 1885, and was the following year elected to the Legislative Council. He later achieved eminence in public and political life and received high honours for his services to the community. Mackellar will perhaps be best remembered for his work on the care of delinquent and handicapped children, which he performed as president of the State Children's Relief Department.



AN

INQUIRY

INTO

THE CAUSES AND EFFECTS

OF

THE VARIOLÆ VACCINÆ,

A DISEASE

DISCOVERED IN SOME OF THE WESTERN COUNTIES OF ENGLAND,

PARTICULARLY

GLOUCESTERSHIRE,

AND KNOWN BY THE NAME OF

THE COW POX.

BY EDWARD JENNER, M. D. F. R. S. &c.

—— QUID NOBIS CERTIUS IPSIS
SENSIBUS ESSE POTEST, QUO VERA AC FALSA NOTEMUS.
LUCRETIUS.

SECOND EDITION.

London:
PRINTED, FOR THE AUTHOR,
BY SAMPSON LOW, NO. 7, BERWICK STREET, SOHO:
AND SOLD BY LAW, AVE-MARIA LANE; AND MURRAY AND HIGHLEY, FLEET STREET.

1800.

3a 134-84

FIGURE I.
Title page of the 1884 Sydney reprint of the second edition of Jenner's "Inquiry".

The third medical name appearing in the preface is that of Dr. John Mildred Creed (1842-1930), editor of *The Australasian Medical Gazette*, honorary surgeon to the

Sydney Hospital and member of the Legislative Council. Creed was a popular and forceful personality, and like Mackellar pursued an active political and public life. He was an assiduous worker for the public health, and is known especially as a pioneer of cremation. Creed's editorial writings included criticisms of the current apathy towards vaccination. The following extract displays his trenchant style:

> The benefits of vaccination being so self-evident that all, except some few persons blinded by prejudice so erroneous, as to lead to the supposition that they are slightly insane; and some others, who become anti-vaccinationists from the conviction that they will obtain notoriety, and the consequent pecuniary advantages . . . renders it unnecessary to discuss the main principles.

It appears probable that the preface to the reprint was from the pen of John Creed.

PREFACE.

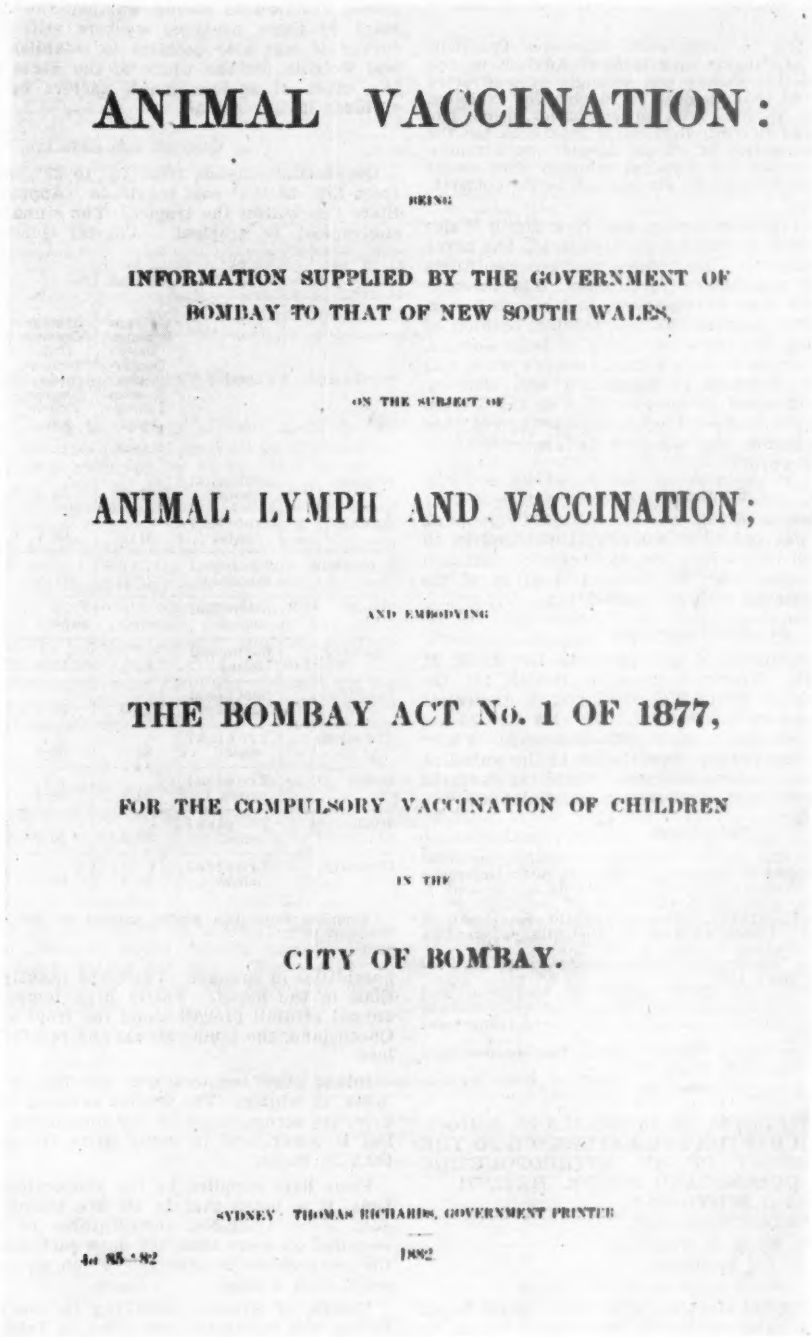
THE original work by Dr. Jenner on the Cow Pox, published in 1799, and a second edition, enlarged, containing all his original observations on the Variola Vaccinæ, in 1800, being now of great rarity,—Dr. G. Bennett having a copy of the second edition of the work, waited, with Dr. Mackellar and Dr. Creed, upon the Hon. A. Stuart (the Premier) to request the Government of New South Wales to reprint the book. This application was granted, and a perfect *fac simile* of the work, in size, type, and the four coloured plates, has been produced. The book will no doubt be found of great benefit to the Medical Profession and the general public, by placing before them, without extraneous matter, the evidence upon which Vaccination was adopted by every civilized Government in the world.

FIGURE II.

Preface of the Sydney reprint of Jenner's "Inquiry". The first edition was actually published in 1798, and not 1799 as shown.

Although the inserted preface to the reprint describes this as "a perfect *fac simile* of the work, in size, type, and the four coloured plates", marked differences occur. The printing is in a later and slightly larger type, in which the old "s" form does not appear. The half-title is omitted, and the title page, though a fairly close reproduction of the original, bears in small type a job number of the

local printer. On the reverse of the title, blank in the original, appears "Reprinted by Authority: Thomas Richards, Government Printer, Sydney, 1884". A preface Observations Relative to the Variolæ Vaccinæ", with its own half-title and title pages, is included. The four coloured engravings are well reproduced in lithograph, the



ANIMAL VACCINATION:

BEING

INFORMATION SUPPLIED BY THE GOVERNMENT OF BOMBAY TO THAT OF NEW SOUTH WALES,

ON THE SUBJECT OF

ANIMAL LYMPH AND VACCINATION;

AND EMBODYING

THE BOMBAY ACT No. 1 OF 1877,

FOR THE COMPULSORY VACCINATION OF CHILDREN

IN THE

CITY OF BOMBAY.

SYDNEY: THOMAS RICHARDS, GOVERNMENT PRINTER

4o 85 '82 1882

FIGURE III.
Title page of a New South Wales Board of Health pamphlet, 1882.

occupying a single page is inserted, and this, with the differences in type, alters the pagination. As in the original, a second part, "A Continuation of Facts and artist's and engraver's names being omitted. The text has been corrected from the *errata* page of the original, which is not included.

The unsuccessful struggle for compulsory vaccination in New South Wales continued for many years after the issue of the reprint. Some twelve years later, in an article which praised the publication, *The Australasian Medical Gazette* (1896) still complained of the absence of legislative action:

> During the last 15 years each successive President of the Board of Health and Medical Adviser to the Government of this Colony has strongly animadverted on the gross and total neglect of vaccination exhibited by the annual returns, and in emphatic terms has earnestly advised the introduction of measures for the compulsory vaccination of all the inhabitants, strongly urging as an excuse the dreadful calamity that would accrue from the widespread devastation by an epidemic of small-pox.

But local politicians were unmoved, and New South Wales remains one of the few British legislatures which has never adopted such a measure. Today, when changed conditions no longer call for compulsory vaccination, this inaction may appear to have been unimportant, and to some even commendable. But it opposed the best medical opinion of the time and flouted the repeated advice of local doctors. For this reason it appears certain that Jenner's work was reprinted for the education of legislators and electors, rather than of the medical profession. For, as the preface states with startling frankness, the book gives "the evidence upon which Vaccination was adopted by every civilized government in the world".

Summary.

The Sydney reprint of the second edition of Jenner's "Inquiry", which was published for official distribution in 1884, was associated with a long and unsuccessful campaign for compulsory vaccination. An account is given of the circumstances connected with its publication.

Acknowledgements.

Grateful acknowledgements are made to Dr. J. H. L. Cumpston, formerly Director-General of Health for the Commonwealth, whose "History of Small-pox in Australia" has been freely used in this compilation. My thanks are also due to Dr. R. Scot Skirving for his assistance. I may be allowed also to express my appreciation of the unfailing kindness, wise counsel and rare humour that have endeared Dr. Scot Skirving to many generations of students.

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HEIGHTS AND WEIGHTS OF QUEENSLAND SCHOOL CHILDREN, WITH PARTICULAR REFERENCE TO THE TROPICS: A REPORT OF AN ANTHROPOMETRIC SURVEY BY QUEENSLAND SCHOOL HEALTH SERVICES.

By P. R. PATRICK,
Brisbane.

IT is now ninety years since the first white people began to settle in those parts of North Queensland which lie within the tropics. This early settlement was accompanied by prophecies of failure which predicted physical deterioration of the race as a result of life in the tropics. Subsequent events have proved that this tropical settlement by whites in Queensland has not been the failure predicted.

The work of Breinl (1921), Cilento (1925), Lee (1940) and Watson-Brown (1938) has proved that it is quite possible for the white man to adapt himself to the climatic conditions found in these parts.

At Professor D. H. K. Lee's suggestion, it was decided that an anthropometric survey should be made amongst school children to decide whether the opinions put forward by these previous workers still held. From the survey it was also possible to establish average heights and weights for the whole of the State and to ascertain the effect of socio-economic factors on the growth of children in Queensland.

QUEENSLAND CLIMATE.

Queensland extends from 10° to 29° south latitude, and from 138° to 154° east longitude. Approximately half the State lies within the tropics. The climate is described as subtropical to tropical. Coastal Queensland has high

TABLE I.[1]

Town.	Locality.	Average Maximum Daily Temperature. (Degrees Fahrenheit.)	Average Minimum Daily Temperature. (Degrees Fahrenheit.)	Average 9 a.m. Relative Humidity. (Percentage.)	Average Annual Rainfall. (Inches.)
Brisbane ..	Subtropical coastal ..	77·9	59·9	67	45
Ipswich ..	Subtropical coastal ..	81·1	56·4	64	33
Toowoomba	Subtropical subcoastal	73·2	51·5	72	36
Roma ..	Subtropical inland ..	82·3	54·5	56	22
Charleville	Subtropical inland ..	84·1	56·0	50	19
Cunnamulla	Subtropical inland ..	82·5	56·8	49	13
Townsville ..	Tropical coastal ..	82·7	69·3	70	45
Cairns ..	Tropical coasta)..	84·5	68·1	73	89
Hughenden ..	Tropical inland ..	89·5	60·7	50	18
Cloncurry ..	Tropical inland ..	90·1	65·8	39	17

[1] Compiled from data kindly supplied by the Meteorological Bureau, Brisbane.

humidities in summer. These are slightly less in the south than in the north. Fairly high temperatures and high annual rainfall prevail along the tropical coast. In South Queensland, the temperatures and rainfall on the coast are less.

Inland, the temperatures are higher in summer and lower in winter. The higher summer temperatures, however, are accompanied by low humidities. Inland the rainfall is lower, and in many parts the annual fall is less than 20 inches.

From data supplied by the Meteorological Bureau, Brisbane, it is noted that in all five inland towns for which data were available, temperatures of over 90° F. are recorded on more than 100 days each year. At Cloncurry, the temperature is over 100° F. on an average of seventy-seven days a year.

Details of climate prevailing in the towns where this survey was conducted are given in Table I. (Figures for Mount Isa were not available).

METHODS USED IN SURVEY.

Measurements were made at schools in six subtropical and five tropical towns, coastal and inland. In the smaller towns all children available were measured, while in the larger cities cross-sections of the school population were

TABLE II.

Average Heights of Boys from the Seven Centres, in Inches.

Age. (Years.)	Brisbane. S.[1]	Ipswich. W.[1]	Toowoomba. S.	South-West Queensland. W.	Townsville. W.	Cairns. S.	North-West Queensland. W.	Average for State.
5	44·15	44·27	43·63	44·23	44·57	43·76	44·47	44·10
6	46·51	46·86	45·89	45·81	46·44	46·07	47·31	46·38
7	48·51	48·43	48·36	48·55	48·32	48·32	48·47	48·43
8	50·84	50·66	50·37	50·49	50·64	50·63	50·55	50·64
9	52·47	52·76	52·69	51·97	52·52	52·55	52·80	52·54
10	54·38	54·77	53·95	54·05	54·41	54·11	54·78	54·32
11	56·09	55·92	56·51	55·71	55·89	55·72	55·64	56·04
12	58·16	58·16	57·99	57·13	58·27	58·01	57·15	58·01
13	60·26	60·46	60·14	60·34	59·93	60·51	60·53	60·27

[1] "S", measured at the end of summer; "W", measured at the end of winter.

taken. Only those children who had lived all their lives in the area were included.

Information regarding the length of time spent in the area by the child and the parents, as well as the occupation of the father, was gathered by means of a *questionnaire*. The children were of European descent, mostly British. The weights were obtained by scales of the beam balance type, which were checked by weights and measures inspectors at each school, and a measuring stick, which was also checked with a standard yard stick from the Weights and Measures Department, was used to obtain the heights. All heights were taken in bare feet, and for the weighing the boys wore trousers only and the girls bloomers and singlets. School sisters, who were given special instructions before commencing, carried out the survey. All measurements were carefully checked during recording, and the compilation of results was carried out by the Government Statistician by the use of a punch card system.

GROWTH FACTORS.

The factors which influence human growth comprise two main groups—namely, those of heredity and those of environment.

Heredity.

In consideration of the effect of heredity on growth, thought must be given to racial factors and to those of the individual stock within the race. Races differ in stature, and certain families within the races are noted for their tall or small stature.

Bean (1928), in a survey of world stature, found that the British and Scandinavians were the tallest races, and that the smallest stature was found amongst the Negrillos of Central Africa and the Aymaras of Central South America. As was mentioned earlier in this report, the children measured were predominantly British, and racial factors would therefore not influence the results. The large numbers of children included would eliminate any individual family influence.

Environment.

The following factors must be included in those classed as environmental: nutritional, climatic, socio-economic, disease, endocrine, pre-natal, emotional. Quite a number of these are interrelated.

Nutritional Factors.

There is no need to elaborate here on the effect of diet on growth or to list the items which are generally included in an adequate diet. With regard to the present survey, it must be pointed out that, owing to the distance from supplies in certain areas, the lack of certain items of diet as milk, fresh fruit and vegetables might be an influence in retarding growth. This availability or otherwise of certain necessary food items is one of several factors which may be classed together as a geographical influence.

In addition to available diet, pure climatic conditions and endemic disease must be considered in this regional influence on growth.

Bean states that tropical jungle life and arctic cold and waste tend to decrease stature, and cites three factors by which certain geographical areas influence growth. These

TABLE III.

Average Weights of Boys from the Seven Centres, in Pounds.

Age. (Years.)	Brisbane. S.[1]	Ipswich. W.[1]	Toowoomba. S.	South-West Queensland. W.	Townsville. W.	Cairns. S.	North-West Queensland. W.	Average for State.
5	44·0	43·75	43·03	44·37	43·95	42·35	43·13	43·55
6	47·97	48·59	48·14	47·07	47·1	46·72	47·67	47·72
7	52·80	52·62	52·13	53·09	51·61	51·31	53·1	52·43
8	59·42	59·67	57·40	58·33	57·33	57·60	56·7	58·30
9	63·88	64·03	64·19	64·55	64·98	62·61	63·52	63·96
10	69·15	70·91	67·58	69·56	68·36	67·39	70·07	68·87
11	75·89	74·99	79·17	75·12	74·82	73·83	71·63	75·83
12	83·91	84·41	84·26	82·55	82·64	84·50	80·66	83·69
13	96·07	95·95	93·37	94·84	88·11	96·57	92·97	94·12

[1] "S", measured at the end of summer; "W", measured at the end of winter.

TABLE IV.

Average Heights of Girls from the Seven Centres, in Inches.

Age. (Years.)	Brisbane. S.[1]	Ipswich. W.[1]	Toowoomba. S.	South-West Queensland. W.	Townsville. W.	Cairns. S.	North-West Queensland. W.	Average for State.
5	43·73	43·77	43·81	43·94	44·04	43·59	43·79	43·79
6	45·97	45·81	46·25	45·84	45·96	45·38	46·13	45·94
7	48·17	48·63	48·26	47·47	48·55	48·08	47·92	48·25
8	50·23	50·80	49·95	49·76	50·31	49·56	50·47	50·18
9	52·46	52·06	52·18	52·32	52·49	52·62	51·26	52·32
10	54·52	54·51	54·52	53·66	55·12	54·11	54·18	54·47
11	56·78	57·17	57·02	55·44	57·79	56·54	55·26	56·87
12	59·22	59·58	59·56	57·68	59·5	58·86	60·01	59·24
13	61·30	60·89	60·67	59·76	61·05	60·73	60·95	60·88

[1] "S", measured at the end of summer; "W", measured at the end of winter.

factors are: the effect of temperature and humidity—that is, pure climatic factors; the prevalence of endemic diseases; and the availability of food. The same writer also points out that the tallest statures are produced by the active life of the temperate zone with its comforts and attendant food supply.

In a comparison between children born in California and those born elsewhere, Lloyd-Jones (1940) cited a "Californian environment" as the reason why the Californian children were superior in stature at every age by about half an inch. The components of this environment were considered to be: increased sunshine, greater variety in diet, more fresh vegetables and fruit, and more hours of outside play, all stemming from the more equable Californian climate.

Meredith (1941), however, discounted somewhat this regional influence, and stated that the average for stature and weight varied but slightly for white boys residing in different parts of the United States.

The details of climate prevailing in the various Queensland towns where the survey was conducted have already been given.

Most workers acknowledge a variation in relation to climatic season. It is generally held that children increase in height more in summer than in winter, while the increase in weight is greater in winter. Watson-Brown in his work amongst western Queensland children found that there was a definite retardation of growth in summer.

It was not found possible to conduct the survey at the same time in all areas, and it is necessary to bear this factor in mind in the interpretation of results. The time of measuring at each centre is indicated in the respective tables.

Disease Factors.

There are certain systemic illnesses such as congenital heart disease, allergy and chronic respiratory infections which retard growth. These conditions influence the growth of individual children, but should not influence the results in a group survey, unless their incidence is higher in certain areas. From school medical examinations there is no evidence of a greater prevalence in any particular area in Queensland.

At one time in North Queensland, two endemic diseases occurred which could influence growth—malaria and ankylostomiasis. These two are now so seldom found in white children that they could not influence any results in an anthropometric survey.

Socio-Economic Factors.

The term "socio-economic", as Meredith points out, is rather a loose one, and includes such factors as diet, housing, personal habits, clothing and selective mating. It is often crudely estimated from the occupation of the father—this method was used here. It is recognized that children of fathers of different occupational groups do vary in height and weight. In this survey, the measurements of children from occupational groups were compared.

Endocrine Factors.

The endocrine glands which influence growth are the pituitary and the thyreoid. Talbot (1947) sums up these influences as follows.

TABLE V.

Average Weights of Girls from the Seven Centres, in Pounds.

Age. (Years.)	Brisbane. S.[1]	Ipswich. W.[1]	Toowoomba. S.	South-West Queensland. W.	Townsville. W.	Cairns. S.	North-West Queensland. W.	Average for State.
5	42·24	42·15	42·63	43·05	41·42	41·47	40·52	42·08
6	47·03	47·21	48·14	47·49	45·37	46·26	46·91	46·91
7	51·93	52·69	52·54	50·6	52·09	51·34	49·82	51·93
8	57·10	59·12	56·28	56·02	55·55	54·83	60·06	56·85
9	64·17	61·56	62·68	67·81	62·94	62·01	61·07	63·20
10	72·01	70·57	71·48	68·92	70·86	67·00	70·58	70·67
11	79·61	85·27	80·31	76·54	80·30	75·94	73·39	79·87
12	90·38	92·15	91·61	84·95	89·44	85·43	91·04	89·82
13	102·33	97·13	99·37	99·19	99·56	97·79	105·12	99·77

[1] "S", measured at the end of summer; "W", measured at the end of winter.

Assuming a healthy organism and an adequate intake of building materials (proteins, minerals, etc.), after the second year or so of life, the child growth promoting agent is the pituitary growth hormone. Thyroid permits or facilitates this action. In adolescent boys, the testicular androgens act as secondary growth-promoting hormones. In addition, they stimulate masculine muscular development.

TABLE VI.

Comparison of Heights and Weights of Boys of Second Generation in Tropics with those of Brisbane and the Whole of the State.

Age. (Yrs.)	Height in Inches.			Weight in Pounds.		
	Boys with Both Parents Born in Tropics.	Brisbane Boys.	Average for State.	Boys with Both Parents Born in Tropics.	Brisbane Boys.	Average for State.
5	44·17	44·15	44·10	42·96	44·00	43·55
6	46·46	46·51	46·38	47·43	47·97	47·72
7	48·18	48·51	48·43	51·16	52·80	52·43
8	50·74	50·84	50·64	58·24	59·42	58·30
9	52·60	52·47	52·54	63·96	63·88	63·96
10	54·23	54·38	54·32	67·77	69·15	68·87
11	55·87	56·09	56·04	73·64	75·89	75·83
12	58·40	58·16	58·01	85·13	83·91	83·69
13	60·37	60·26	60·27	91·46	96·07	94·12

There is a marked acceleration of growth just prior to or during puberty with a very slow increase in the post-pubertal years. Ellis (1946) states that the more mature boys are significantly taller and heavier than boys in the same age group who have not reached puberty. He further states that this difference could be demonstrated as far back as the sixth year. In a similar study with reference to girls, Wilson and Sutherland (1949) confirmed this concept. Girls who had not started menstruating were considerably shorter and lighter than their fellows who had reached puberty.

TABLE VII.

Comparison of Heights and Weights of Girls of Second Generation in Tropics with those of Brisbane Girls and the Whole State.

Age. (Yrs.)	Height in Inches.			Weight in Pounds.		
	Girls with Both Parents Born in Tropics.	Brisbane Girls.	Average for State.	Girls with Both Parents Born in Tropics.	Brisbane Girls.	Average for State.
5	43·78	43·73	43·79	41·07	42·24	42·08
6	45·77	45·97	45·94	45·83	47·03	46·91
7	48·42	48·17	48·25	51·35	51·93	51·93
8	49·98	50·23	50·18	54·22	57·10	56·85
9	52·34	52·46	52·32	61·27	64·17	63·20
10	54·83	54·52	54·47	69·93	72·01	70·67
11	57·28	56·78	56·87	77·86	79·61	79·87
12	59·35	59·22	59·24	89·34	90·38	89·82
13	60·92	61·30	60·88	98·64	102·33	99·77

This influence of puberty on growth would need to be considered in the present survey if there was evidence that children reached maturity earlier in one region than in another. Information regarding the age of puberty in different parts of the State is incomplete. In the evidence available, there is nothing to support a popular belief that puberty is reached at an earlier age in the tropics.

TABLE VIII.

Average Heights and Weights of Queensland Girls and Boys.

Age. (Yrs.)	Boys.			Girls.		
	Number Measured.	Height. (Inches.)	Weight. (Pounds.)	Number Measured.	Height. (Inches.)	Weight. (Pounds.)
5	605	44·10	43·55	548	43·79	42·08
6	624	46·38	47·72	657	45·94	46·91
7	566	48·43	52·43	571	48·25	51·93
8	604	50·64	58·30	603	50·18	56·85
9	563	52·54	63·96	544	52·32	63·20
10	538	54·32	68·87	556	54·47	70·67
11	567	56·04	75·83	507	56·87	79·87
12	473	58·01	83·69	485	59·24	89·82
13	455	60·27	94·12	470	60·88	99·77
14	160	62·88	105·72	137	62·34	106·76

Cilento found the age of menarche to be the fourteenth year in North Queensland. Watson-Brown found the average age of the onset of menstruation among 500 girls in north-western and central western Queensland to be 13·1 years. These figures are not far removed from the median age of 13·6 years amongst English girls as stated by Wilson and Sutherland.

TABLE IX.

Comparison of Heights and Weights of Queensland Boys with those of Other Countries.[1]

Age. (Yrs.)	Height in Inches, Weight in Pounds.	Queensland, 1950.	New South Wales Amended Table, 1937.	Victoria, 1937–1938, Nutrition Survey—Good Suburban.	America, 1945.	County of London, 1938.
5	Height..	44·10	43·6	44·8	44·1	43·0
	Weight	43·55	42·9	45·7	43·2	42·7
6	Height..	46·38	45·8	46·8	46·4	45·2
	Weight	47·72	47·4	50·2	47·8	47·2
7	Height..	48·43	48·1	49·0	48·5	47·4
	Weight	52·43	51·7	53·4	52·6	52·1
8	Height..	50·64	50·1	51·4	50·7	49·5
	Weight	58·30	57·2	59·4	58·3	57·5
9	Height..	52·54	52·1	53·0	52·7	51·5
	Weight	63·96	62·0	66·6	64·3	63·2
10	Height..	54·32	54·4	54·9	54·5	53·5
	Weight	68·87	68·3	74·0	70·4	69·3
11	Height..	56·04	55·6	56·6	56·3	55·3
	Weight	75·83	74·3	79·7	76·9	75·9
12	Height..	58·01	57·8	57·5	58·3	57·0
	Weight	83·69	81·0	84·5	84·9	83·0
13	Height..	60·27	60·0	60·5	60·7	58·7
	Weight	94·12	91·9	95·0	95·1	90·5
14	Height..	62·88	62·8	—	63·1	—
	Weight	105·72	104·4	—	106·9	—

[1] New South Wales figures taken from table supplied by Director, School Medical Services, New South Wales. Victorian figures from table supplied by Chief Medical Inspector of Schools, Victoria. American figures are those published in "Textbook of Pediatrics" (Mitchell-Nelson), Fourth Edition, 1945, adapted from R. O'Brien, M. A. Girshick and E. P. Hunt, United States Department of Agriculture Publication Number 366. London figures are taken from Sir Frederick Menzies's London County Council Report of 1940.

TABLE X.

Comparison of Heights and Weights of Brisbane Children in 1911 and 1950.

Age. (Years.)	Boys.				Girls.			
	Height. (Inches.)		Weight. (Pounds.)		Height. (Inches.)		Weight. (Pounds.)	
	1950	1911	1950	1911	1950	1911	1950	1911
7	48·51	45·9	52·80	46·5	48·17	45·5	51·93	45·1
8	50·84	48·4	59·42	53·0	50·23	48·0	57·10	50·5
9	52·47	49·8	63·88	56·0	52·46	49·6	64·17	55·5
10	54·38	52·2	69·15	61·00	54·52	51·6	72·01	60·0
11	56·09	53·4	75·89	66·0	56·78	53·7	79·61	67·0
12	58·16	54·8	83·91	71·5	59·22	56·4	90·38	76·2
13	60·26	56·6	96·07	78·5	61·30	57·6	102·33	83·5

Pre-Natal Factors.

From the work of Illingworth (1949) and of Drillien (1948), it appears that there is a constant relation between birth weight and subsequent physical development. Many factors, both hereditary and environmental, influence birth weight.

Drillien points out that the same adverse environmental conditions which are acting during pregnancy are still present in the household during further development after birth. As Illingworth points out, there is need for further investigation on this question.

Socio-Emotional Factors.

All factors considered so far may be termed physical, and most growth studies concern themselves with these physical influences only. A report from Fried and Mayer (1948) shows that socio-emotional states influence both individual health and physical well-being. These workers state that growth failure due to socio-emotional disturbance occurs more frequently and is greater than is generally recognized. In this survey, it is held that these effects would be eliminated by the number of children measured.

Results of Survey.

The figures from the eleven towns where the survey was made were recorded in seven groups. They were: Brisbane, Ipswich, Toowoomba and south-west Queensland (which included Roma, Charleville and Cunnamulla) in the south; Townsville, Cairns and north-west Queensland (which included Hughenden, Cloncurry and Mount Isa) in the north.

From all seven centres, the figures were fairly uniform. In no centre did the heights and weights of both boys and girls vary greatly from the average for the whole of the State. In two groups, Cairns and south-west Queensland, the girls only were below average at most ages for both height and weight. In the figures for the boys from these two areas, there was no significant change from the average. The greatest variation from the average was found in the heights of south-west Queensland girls, who, in the eleven, twelve and thirteen years age groups, were more than one inch shorter than the average.

The variation in weight was not so significant. An analysis of the figures showed that this difference from average was present in all three towns which formed the group. Later on it will be shown that height has a definite relationship to the father's occupation; but the percentage of the various occupations did not vary in south-west Queensland from those in other centres.

The numbers of girls measured in south-west Queensland are small, but all those available were included. The measurements were made at the end of winter, when the rate of height increase is generally considered to be the lowest; but when comparisons are made with the other centres measured at the same time, there is still a considerable difference.

This isolated instance of shortness in a small number of age groups of one sex only is not sufficient evidence from which to conclude that geographical region has a pronounced influence on stature.

The Cairns girls, while shorter and lighter than the average, were not noticeably so. The greatest difference in height was approximately half an inch and that in weight was four pounds; but the difference in other age groups was much less, and thirteen-year-old girls in this centre were only 0·15 inch shorter and two pounds lighter than the average. This variation may be explained by seasonal growth, as the measurements in Cairns were taken at the end of summer. The survey was also carried out at this time in Brisbane and Toowoomba, where the results were generally above average. However, the tropical summer may have a greater effect on growth than that of southern Queensland. It is hoped to clear up this point by a further longitudinal growth study in both South and North Queensland. The results for all centres are recorded in Tables II to V.

The Relation of the Tropics to Height and Weight.

Previous workers in Queensland have stated that life in tropical Queensland has not had an adverse influence on the physique of children. Cilento stated that figures obtained after examining 2080 children "proved conclusively that there is no appreciable difference in the mental and physical development of the children born within the tropics, and the immigrants".

The same writer also found that "Townsville children were several pounds lighter than the children in other towns, and that the height at all ages, and in both sexes, varies from one to three inches above normal in all the towns except Charters Towers". The discrepancy in weight at Townsville was explained by the fact that the children were weighed at the end of summer.

Weights in the Tropics.

In the present survey, the weights of children at Townsville (measured at the end of winter) varied no more than one pound from the average in all age groups of both sexes, except those of the thirteen-year-old boys.

The weights of Cairns children weighed at the end of summer were not much at variance with the average. The boys were one to two pounds below average weight in the lower age groups, but at twelve years and thirteen years they were heavier. The Cairns girls, as mentioned earlier, were slightly lighter in all age groups.

In north-west Queensland, the boys were lighter than average, but not to any great extent, and the thirteen-year-old boys were only one pound lighter than average. The weights of north-western Queensland girls, while varying more from the average in different age groups, were on the whole up to the average.

Heights in the Tropics.

The heights of children living in tropical areas compare favourably with those of children in other centres. In two age groups in the north-western girls, the figure was more than one inch below average; but this was compensated for by measurements above average in other age groups.

The Townsville girls were above average height, but the difference was not significant.

The slight difference below average of the Cairns girls has already been mentioned. On the whole, the boys in the northern centres followed the average fairly closely.

TABLE XI.

Average Heights and Weights of Boys According to Father's Occupation.

Age. (Yrs.)	Height in Inches, Weight in Pounds.	Rural, Fishing and Hunting.	Professional and Semi-Professional.	Administrative, Commercial and Clerical.	Craftsmen and Operatives.	Labourers.
5	Height..	44·13	45·21	44·62	43·93	43·79
	Weight	43·98	45·93	44·53	43·25	42·81
6	Height..	46·50	46·34	46·66	46·32	45·90
	Weight	48·46	47·06	48·45	47·53	46·54
7	Height..	48·36	48·85	48·97	48·25	48·18
	Weight	52·99	54·12	53·54	52·11	51·77
8	Height..	50·59	51·53	51·05	50·60	50·20
	Weight	57·19	58·97	60·28	58·29	56·49
9	Height..	52·18	53·24	52·64	52·52	52·35
	Weight	63·12	66·26	63·75	64·41	62·27
10	Height..	53·97	55·27	54·40	54·68	53·92
	Weight	69·75	73·22	69·34	69·73	66·77
11	Height..	55·89	57·41	56·13	55·99	55·60
	Weight	75·05	80·34	76·07	75·19	73·08
12	Height..	58·35	58·21	58·18	58·01	57·38
	Weight	85·99	83·94	83·38	83·64	82·42
13	Height..	60·69	61·25	60·89	60·16	59·44
	Weight	93·67	94·26	98·87	93·52	91·06

Generation Grouping.

From information received during the survey, it is possible to group children according to generations. In Tables VI and VII measurements for children of the second generation in Townsville and Cairns are compared with averages for Brisbane and the State. In this composite tropical group, the Townsville children were weighed at the end of winter, and the Cairns children at the end of summer, and the seasonal effect would be practically eliminated.

In most age groups, both sexes approximated the average height, but were a little below average weight. The differences in weight, however, are very small, and are not progressive from year to year, so that it may be safely said that after two generations in the tropics, the height and weight of children have been unaltered.

The eight-year-old and nine-year-old boys could be taken as truly representative of Queensland child growth, so closely do their measurements resemble the average for Queensland.

These results confirm the statements of previous workers that the physique of children living in tropical Queensland has not deteriorated by their sojourn in those areas; but they do not support the statement that children in northern towns are one to three inches taller than normal.

State Averages.

Queensland average heights and weights for age are given in Table VIII. The heights and weights of Queensland boys are compared in Table IX with those for New South Wales, Victoria, England and America. Such comparisons must be made with caution, consideration being given to the fact that methods used, clothes worn, date of survey *et cetera* vary. As the figures stand, Queensland children compare favourably with other children.

Secular Change in Growth.

Throughout the world there has been an increase in stature over the last fifty years. Menzies (1940) found "the height of a child 5½ years of age today (1938) is much the same as that of a child 6½ in 1905-1912, i.e. the increase both in height and weight during the period is approximately equal to one year's growth". Daley in 1947 found that the growth increase in English children was continuing, there being a further increase of one inch in height at all ages between 1938 and 1947.

TABLE XII.

Average Heights and Weights of Girls According to Father's Occupation.

Age. (Yrs.)	Height in Inches, Weight in Pounds.	Rural, Fishing and Hunting.	Professional and Semi-Professional.	Administrative, Commercial and Clerical.	Craftsmen and Operatives.	Labourers.
5	Height..	43·26	43·86	43·89	43·90	43·37
	Weight	43·55	42·80	42·23	42·32	40·35
6	Height..	45·95	46·02	45·79	46·05	45·61
	Weight	47·73	47·35	46·82	47·05	45·28
7	Height..	48·06	49·99	48·46	48·63	47·69
	Weight	52·03	56·48	51·70	52·15	50·48
8	Height..	49·72	50·47	50·33	50·22	49·65
	Weight	55·58	56·78	57·41	57·01	54·72
9	Height..	52·26	52·26	52·65	52·24	51·97
	Weight	62·07	65·65	64·19	63·87	60·02
10	Height..	54·5	55·72	54·98	54·38	53·59
	Weight	71·0	75·03	74·94	70·18	66·43
11	Height..	56·54	57·18	57·07	56·8	56·58
	Weight	79·93	86·19	81·26	79·15	79·12
12	Height..	59·70	60·35	59·39	59·26	59·17
	Weight	89·28	103·83[1]	90·52	90·58	89·37
13	Height..	61·49	61·20	61·13	60·86	60·40
	Weight	103·18	98·76	101·88	98·64	98·92

[1] The apparent discrepancy in the weight of twelve-year-old girls from the professional class is due to the low numbers measured in this particular age group.

TABLE XIII.

Annual Increases in Heights and Weights.

Age Range in Years.	Girls.		Boys.	
	Heights in Inches.	Weights in Pounds.	Heights in Inches.	Weights in Pounds.
5½ to 6½ ..	2·15	4·83	2·28	4·17
6½ to 7½ ..	2·31	5·02	2·05	4·71
7½ to 8½ ..	1·93	4·92	2·21	5·87
8½ to 9½ ..	2·14	6·35	1·90	5·66
9½ to 10½ ..	2·15	7·47	1·78	4·91
10½ to 11½ ..	2·4	9·2	1·72	6·96
11½ to 12½ ..	2·37	9·95	1·97	7·86
12½ to 13½ ..	1·64	9·95	2·26	10·43
13½ to 14½ ..	1·46	6·99	2·61	11·60

In 1911 Bourne weighed and measured children in Brisbane schools; these children are compared with those of 1950 Brisbane children in Table X, and it will be seen that this secular change is occurring here also—in every age group there is a considerable increase.

The Effect of the Father's Occupation.

The figures from the whole State were grouped according to the occupation of the father in Tables XI and XII. At all ages in both sexes, the children of professional fathers were taller and heavier than those whose fathers were labourers; generally children from the professional and

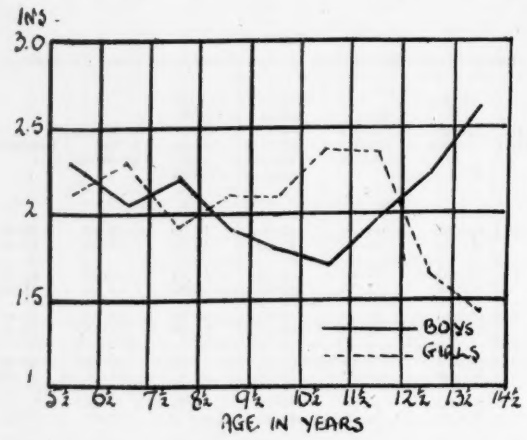

FIGURE I.

Graph comparing the annual increase in heights of boys and girls. Note the fairly even rate from five and a half to eight and a half years in both sexes, the comparatively slow rate in boys between eight and a half and eleven and a half years, and the earlier spurt and earlier lessening of the rate in girls. In boys, the fastest increase is in the fourteenth year, when girls are slowing.

administrative classes were taller and heavier than those from the craftsmen and labourer classes. Children from rural occupational groups occupied a central position between these two main divisions.

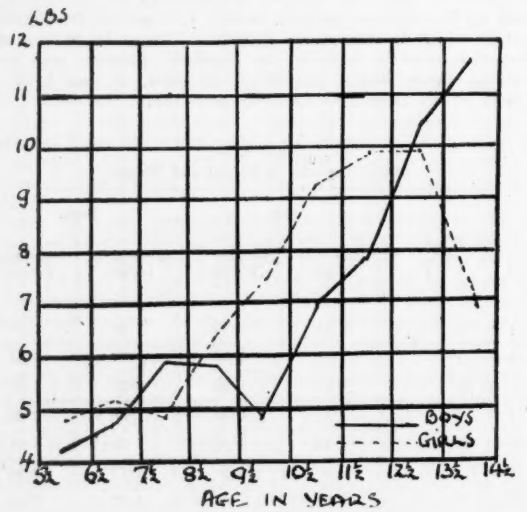

FIGURE II.

Graph comparing the annual increase in weight of boys and girls. Again note the earlier spurt and earlier lessening of the rate in girls.

The difference in heights between children from professional occupational groups and those from the labourer groups, although it varies from one age group to another, seems to be progressive. The advantage in height, which is apparent in both sexes at five years, is increased a further half an inch during growth from five years to thirteen years.

The difference in weight, which is present at all ages, does not increase proportionately with age, and both boys and girls in the upper age groups of all occupational classes do not vary significantly in weight.

The occupation of the father has more significance, at least as regards height, than has geographical region.

Annual Rates of Growth.

The average annual rates of growth are given in Table XIII and in Figures I and II.

In the boys, the annual increment in height is over two inches from five and a half years to eight and a half years. There is then a decrease in the growth rate, which is slowest between nine and a half and eleven and a half years. From that age on, there is an increase in the growth rate. This is greatest between thirteen and a half and fourteen and a half years, when the annual rate is the highest of any stage under review. While the annual weight increase did vary a little from the five and a half

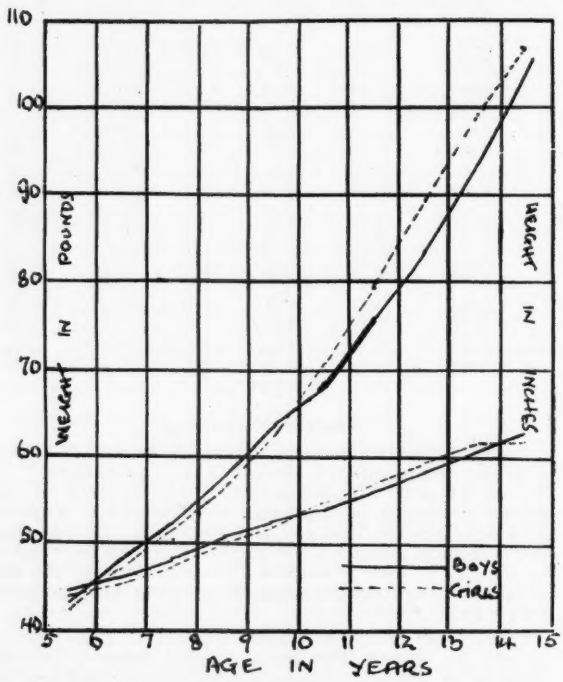

FIGURE III.

Graphs comparing average heights and weights for age of Queensland girls and boys. Note that from five years to ten years, boys are slightly taller and heavier than girls. From ten years to fourteen years the position is reversed, but during the fifteenth year the boys have again become taller and are approximating the girls in weight.

years to ten and a half years group, it was fairly constant. From then on, there is increase in the annual rate, and this is greatest between thirteen and a half and fourteen and a half years.

The girls increased in height at a rate of about two inches a year until ten and a half years. From ten and a half years to twelve and a half years, there was a spurt in height, but not as much as among the boys, and the spurt was not maintained for so long a period. From twelve and a half years to fourteen and a half years, the annual rate of increase in height was rapidly decreasing.

From five and a half years to eight and a half years, there was an annual increase in the weight of girls of approximately five pounds. From then until thirteen and

a half years, the annual rate quickened. From ten and a half years to thirteen and a half years, it was over nine pounds annually. In the next year, the yearly rate of increase was slowing up.

Up to approximately ten years, the boys were taller and heavier than the girls. At that age, the girls began to forge ahead, and for about four years were taller and heavier. This spurt was not maintained so long as that of the boys, and at approximately fourteen years the boys had equalled the girls in height and were catching them in weight.

The average heights and weights of boys and girls are compared in Figure III.

CONCLUSION.

1. This work supports the opinions of earlier workers that the heights and weights of children living in tropical Queensland vary little from those of children in other parts of the State.

2. Queensland children compare favourably in heights and weights with children from other countries.

3. The occupation of the father bears a definite relationship to the height of children in Queensland.

4. There has been an increase in height and weight in Brisbane children over the last forty years comparable with secular changes found elsewhere.

5. Up to approximately ten years of age, Queensland boys are slightly taller and heavier than Queensland girls. From ten years to fourteen years, the position is reversed.

ACKNOWLEDGEMENTS.

I am indebted to Dr. A. Fryberg, Director-General of Health and Medical Services, Department of Health and Home Affairs, Brisbane, for permission to publish this report.

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Reports of Cases.

ACQUIRED HÆMATOMETRA: REPORT OF TWO CASES.[1]

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Case I: Hæmatometra following a Difficult Confinement.

MRS. B., aged thirty-five years, nulliparous, gave a past history of having had five abortions. Appendicectomy had been performed one year prior to confinement. Her confinement took place in July, 1947. Antenatal supervision had been carried out and the course of pregnancy was normal.

The details of a long and difficult confinement may be summarized as follows. Labour commenced some three weeks after the expected date of delivery, with the fœtal head engaging in the right occipito-posterior position. Primary uterine inertia was a feature of the labour and was treated along routine lines. The membranes ruptured prematurely after sixty hours of labour while vaginal examination and rotation of the head were being performed. After a labour of 106 hours a stillborn child weighing ten pounds six and a half ounces was delivered by difficult forceps extraction. The placenta was adherent and manual removal was performed one and a half hours after the birth of the baby. Glucose and saline solution were given intravenously followed by blood transfusion, and perineal and vaginal lacerations were sutured. During labour the patient had three mild rigors and her temperature was elevated to 101° F. on these occasions only. Penicillin, sulphonamide drugs and gas-gangrene antiserum were given after delivery. The puerperium was afebrile and the patient was allowed out of bed on the tenth day and was discharged from hospital on the fourteenth day after confinement.

Routine postnatal examination was performed five weeks after confinement (August 12, 1947). The patient then complained of frequency of micturition (six to eight times per day, two or three times at night), stress incontinence and low backache. Abdominal findings were negative and vaginal examination revealed a lax pelvic outlet with cystocele and rectocele present. No other relevant abnormality was noted. The patient reported three months later (November 18, 1947), complaining bitterly of inability to control the urine. The urine escaped when she was standing, walking or coughing. The menstrual periods had not returned and she had dull pain in the left iliac fossa associated with nausea. Pregnancy could have been possible six weeks after confinement—that is, in early September. Catheterization after the passage of urine yielded one ounce of residual urine, and a mass could be felt in the lower part of the abdomen, reaching half-way to the level of the umbilicus. On pelvic examination the cervix could not be felt or seen and the site of the external os was not apparent. A globular cystic mass was felt through the vaginal vault, which had the characteristics of a sixteen weeks' pregnancy. The patient was examined in consultation by a senior colleague, who thought he could detect the obliterated cervix but was not sure. He considered the condition probably a uterine pregnancy and advised Cæsarean section near term preceded by examination under anæsthesia to make sure of the presence of the os. Severe incontinence of urine was still present. Fifteen days later (December 21, 1947) the patient developed acute retention of urine. Forty ounces of urine were withdrawn by catheter and she was admitted to hospital at once. Routine catheterization was performed in hospital, and X-ray examination revealed no evidence of fœtal parts. On the second day in hospital, while on the

[1] Read at a meeting of the Section of Obstetrics and Gynæcology of the New South Wales Branch of the British Medical Association on April 3, 1951, at Sydney Hospital.

bed-pan, the patient passed a large quantity of gluey chocolate-coloured blood and the diagnosis of hæmatometra was established. Examination of the patient under anæsthesia revealed a small ragged opening in the region of the cervix, which admitted a fingertip. Dark blood oozed from this opening. The uterus was soft and enlarged to the size of a fourteen weeks' pregnancy and the fornices felt clear. The cervix was slowly dilated to admit size 16–19 Hegar's dilator, and a hot intrauterine douche was given. The urinary symptoms disappeared immediately and convalescence was uneventful. She was discharged from hospital four days later. When examined three weeks subsequently the patient felt well and complained only of mild frequency of micturition and very occasional stress incontinence of urine. She had menstruated from January 13 to 16, and the cervix appeared normal.

On February 17, 1948, she reported with continuous hæmorrhage since January 30, with several heavy floodings during the past two weeks. Pelvic examination was attempted, but severe vaginal bleeding occurred and the patient was again promptly admitted to hospital. Examination in the operating theatre revealed a large distended cervix with a small os from which dark blood escaped. The uterus was enlarged and felt soft. Dilatation of the cervix was again performed up to size 17–20 Hegar's dilator, and thick dark blood was washed from the uterus. Pelvic examination four days later showed that the os admitted a finger tip and the uterus was enlarged to the size of an eight weeks' pregnancy and anteverted. She was instructed to report if untoward symptoms developed, but was not examined until nine months later (September 30, 1948). The menstrual periods had been fairly regular; there was still some frequency of micturition, but urinary control was good. The cervix felt and looked normal, the condition of vaginal prolapse had decreased and the uterus was anteverted and normal in size.

The patient was examined three and a half months later (January 13, 1949) with a history of two missed menstrual periods, the last menstrual period having been from November 7 to 11, 1948. Examination established the diagnosis of an eight weeks' uterine pregnancy. The antenatal course was normal and a baby weighing eight pounds eleven and a half ounces was delivered by lower segment Cæsarean section ten days before term. The puerperium was uncomplicated and the patient has had no further trouble.

Case II: Hæmatometra of the Residual Cervix.

On July 12, 1950, Mrs. P., aged forty-seven years, gave a history of having had one child twenty-two years earlier. The pregnancy was complicated by preeclamptic toxæmia, the labour was difficult and stitches were necessary. There was alleged "heart trouble" following the confinement and the patient was in hospital for about three months. The operations of amputation of the cervix, subtotal hysterectomy and reaming out of the cervical canal had been performed at the Royal Newcastle Hospital in December, 1939.

While doing housework on July 1, 1950, the patient noticed a sudden gush of much "tarry material" from the vagina, and one week later she passed another cupful of this dark fluid. Eighteen months earlier she had had a scanty brown discharge of short duration. Prior to passing the dark material on July 1 the abdomen felt distended and uncomfortable, and she felt a "lump" in the left lower quadrant of the abdomen. She stated that the lump disappeared after she had passed the dark fluid. Other symptoms before the vaginal bleeding occurred were severe constipation, lumbar backache and slight swelling of the left lower limb. Her doctor considered the condition probably due to the rupture of an ovarian cyst into the vagina.

On examination the patient was found to be a fairly healthy middle-aged woman. A grid-iron appendicectomy scar and a left paramedian section scar were present on the abdomen. The abdominal wall was lax and localized tenderness was present over the left adnexa with vague tenderness in the left hypochondriac area. No guarding or mass could be felt in the abdomen. Vaginal examination revealed a repaired perineum, with no prolapse, no bleeding and no discharge. The residual cervix was felt high in the vaginal vault and was enlarged in its supravaginal segment chiefly towards the left side. No other pelvic mass was felt. A small laceration was present at the region of the external os, but the patency of the os was not tested by the passage of a sound or probe. Rectal examination revealed no abnormality except external hæmorrhoids. A provisional diagnosis was made of possible hæmatometra of the residual cervix, or possible chocolate cyst of the ovary discharging through the residual cervix.

At operation on September 15, 1950, after vaginal toilet and catheterization the cervix was inspected. The site of the os was evident, but it was found to be stenosed so that a fine probe could not be passed into the endocervical canal. In view of doubt concerning other pelvic or abdominal disease section was performed. After a few adhesions had been separated the supravaginal part of the cervix was identified and found to be enlarged to half the size of a normal uterus. The right ovary was sclerosed and a little enlarged and the left ovary and appendix had been removed at previous operations. Examination of other abdominal organs revealed no abnormality. The right ovary was removed and the enlarged residual cervix was totally removed. When the dissection reached the region of the vaginal vault some dark chocolate-coloured blood escaped from a small hole made in the attenuated wall of the cervix. Convalescence was straightforward and the patient left hospital on the fifteenth post-operative day. When subsequently examined on November 22, 1950, she looked much better and had gained weight. There were no pelvic signs or symptoms and examination revealed no abnormality.

A pathological report made by Dr. A. A. Palmer, of Sydney Hospital, on October 4, 1950, was as follows:

> The following specimens were received:
>
> 1. A cylindrical piece of firm tissue 4·5 cms. long and 2·5 cms. in average diameter with ragged surfaces except at one end which was covered with mucosa. The specimen appeared to be the cervix. There was a central cyst filled with gelatinous material 1·0 cm. in average diameter. A tract led to the upper end of the specimen, but the external os appeared to be occluded. The cyst was surrounded by brown staining suggesting old blood pigment.
>
> 2. An ovary measuring 3 cm. × 2·0 cm. × 1·5 cm. containing two fairly recent *corpora lutea*.
>
> Microscopic examination: Sections confirm the above description. There is squamous metaplasia of part of the epithelium lining the cyst which appears to be the occluded cervical canal and contains some old blood.

Discussion.

Obstruction of the menses has been recognized since the time of Celsus; but the first recorded case of hæmatometra following difficult delivery is probably that of Dance in 1829. In view of the variety of forms of gynatresia which may occur in the lower part of the genital tract, and considering the possibilities of acquired obstruction of the cervix due to trauma, disease or cicatricial contraction, the condition of hæmatometra is not so common as might be expected. Something over 200 cases of hæmatometra had been reported in the literature up to the year 1947. Bernstein and Walter reported 19 cases—11 congenital and eight acquired—at the Mount Sinai Hospital over the twenty-year period from 1917 to 1927, and estimated the incidence at about 1 in 1000 gynæcological hospital admissions. Simon reported 23 cases from the Mayo Clinic—11 congenital and 12 acquired—over a fifteen-year period from 1912 to 1926.

Two conditions are necessary for the development of hæmatometra—a uterus or part of a uterus capable of menstruating and some type of obstruction preventing the escape of the menses. It may be noted, however, that hæmatometra has been reported in the newly born, associated with endocrine anomalies, and in post-menopausal women owing to neoplasm or to the indiscriminate use of œstrogens in women who have a stenosed cervix.

Congenital causes have been considered to outnumber acquired causes; but nowadays an increased incidence of

cervical stenosis follows operations on the cervix. Melody considers that less than 10% of cases of cervical obstruction are congenital in origin.

The site of obstruction in acquired hæmatometra is usually the cervix, susceptible as it is to trauma and infection at childbirth and miscarriage, and to inflammation, neoplasia and stenosis following surgical procedures. Cauterization of the endocervical canal is now considered to be the most important single cause of stenosis due to trauma, and an incidence of about 9% of cervical stenosis is reported after conization of the cervix (Melody). Hæmatometra is rarely encountered after amputation of the cervix and does not appear to be a late complication of the Manchester operation. Acquired hæmatometra has been reported after local caustic applications to the cervix and following the insertion of radium for menopausal bleeding. Other causes are neoplasia of the cervix, simple and malignant, and *fibromyomata uteri* obstructing the lumen of the cervical canal. Rare miscellaneous causes of acquired hæmatometra include the wearing of stem pessaries, cervical ulceration in prolapse cases, gonorrhœal and diabetic vaginitis and syphilitic stenosis.

The altered incidence between post-partum cases and post-operative cases has a parallel in the altered incidence of vesico-vaginal and recto-vaginal fistulæ. The latter were more common after difficult confinement, but are now seen chiefly after pelvic operations and radium treatment. Were it not for the more important consideration of cancer prophylaxis, it might appear that the improved results of modern obstetrical practice are to some extent offset by too vigorous surgical treatment of the *cervix uteri*.

Injury of the cervix at childbirth accounted for three of the 12 cases of acquired hæmatometra reported by Simon and for three out of eight cases in Bernstein and Walter's series. Tait reported a case of hæmatometra due to cicatricial stenosis of the cervix seven months after difficult labour, and Allen reported a similar case. The history of difficult confinement in the first case reported bears a striking resemblance to the cases of Tait and Allen. There is evidenec in the literature that confinements prone to be followed by hæmatometra follow a close pattern. Post-maturity, long and difficult labours, early rupture of the membranes, difficult forceps extraction, manual removal of the placenta and puerperal infection are characteristics of this pattern.

Hæmatometra of the residual cervix is a rare condition. Hallendorf and Lovelace reported a case of hæmatocolpometra nineteen years after subtotal hysterectomy. Pyometra of the residual cervix is not rare. The diminished blood supply to the cervix and contraction of the endocervical canal dispose to stricture formation. Scanty periodic bleeding from the cervical stump is occasionally encountered and necessitates routine investigation for carcinoma. Henriksen traced the records of 6550 patients at the Johns Hopkins Hospital who had undergone subtotal hysterectomy for benign uterine disease. Of those patients who returned for treatment because of complaints relative to the cervical stump, 36% had periodic vaginal bleeding. One patient had carcinoma and the remaining 56 patients had functioning endometrium. Hæmatometra of the residual cervix might be expected to follow high amputation of the corpus, active endometrium being left, associated with some factor disposing to stenosis of the cervical canal. In the second case cited, hæmatometra developed eleven years after amputation of the cervix, subtotal hysterectomy and reaming the cervical canal.

Pain was not an important feature of the first case and urinary symptoms predominated. The second patient had more pain, chiefly in the left abdominal and left lumbar regions. Abdominal pain of a cyclic nature is not such a characteristic of acquired hæmatometra as it is of congenital hæmatometra. There is usually no associated hæmatocolpos, stenosis is frequently incomplete and the pelvic mass is usually smaller. Moreover, the uterus at this time of life is physiologically adapted to progressive painless enlargement. The body of the uterus is essentially a muscular organ capable of distension, while the cervix consists chiefly of fibrous tissue.

Bleeding was not present in the first case until spontaneous evacuation of the hæmatometra occurred. Spontaneous evacuation of the hæmatometra usually occurs through the site of obstruction at the cervix, but rupture into the peritoneal cavity, bladder, rectum and labia has been reported (Simon). The recurrence of incomplete obstruction two months later was signalled by the irregular passage of dark blood, and a severe hæmorrhage occurred when vaginal examination was attempted. In Case II the patient had two epochs in which dark blood was passed eleven years after operation.

Amenorrhœa of five months' duration from the time of confinement was present in Case I; in Allen's case the diagnosis was made nine months after delivery, and in Tait's case seven months after delivery.

Urinary symptoms were outstanding in the first case. Stress incontinence with associated cystocele was, no doubt, mistakenly diagnosed for retention of urine with overflow incontinence. Acute retention of urine precipitated the admission of the patient to hospital. Urinary symptoms in the form of frequency of micturition, dysuria, overflow incontinence or complete retention of urine are more common in congenital hæmatometra. Trafton and Ewert consider that these urinary symptoms are present in 30% of cases of hæmatometra.

The diagnosis of acquired hæmatometra presents more difficulties than that of congenital hæmatometra, and correct pre-operative diagnosis is the exception rather than the rule. Pregnancy in a retroverted uterus is the common diagnostic pitfall, and was the mistaken diagnosis in Case I. Investigation by one of the biological tests of pregnancy would have facilitated diagnosis. In view of the symptomatology discussed—pain, bleeding, amenorrhœa and urinary symptoms—it is apparent that the diagnosis of hæmatometra can be confused with that of abortion or ectopic pregnancy.

Treatment of the first patient by dilatation and irrigation was followed by a recurrence of hæmatometra, but further similar treatment was effective and a successful pregnancy followed. Simon has observed that in untreated cases with spontaneous rupture recurrence and possibly pyometra usually follow. The vaginal route is preferred for routine removal of the residual cervix for benign lesions, but the possibility of other abnormality influenced the performance of section in the second case. The residual cervix which bleeds is to be viewed with suspicion even when malignant disease is not proven, and is preferably treated by the safe and simple operation of removal rather than by conservative methods. The evolution of the surgical treatment of hæmatometra from blind drainage with a trochar to modern conservative plastic surgery is of interest. The age and parity of the patient and the individual problem involved are to be considered. Women who wish to retain the functions of menstruation and child-bearing are not submitted to hysterectomy unless conservative treatment has failed. Vaginal drainage of a hæmatometra with hæmatosalpinx has been considered dangerous without preliminary section to ascertain the condition of the Fallopian tubes. It would appear that the risk of the tube's rupturing during vaginal drainage has been exaggerated. When gradual dilatation of the stenosed cervix is impossible, anterior lip bisection can be performed. Allen's case is an example of conservative treatment of a hæmatometra with hæmatosalpinx with complete obliteration of communication between the uterus and vagina. The uterus was opened and evacuated and a new communication was made with the vagina by a plastic operation over a tube. Even complex anomalies found in congenital hæmatometra have been successfully treated without removal of the uterus, as in the case reported by Bonney and McIndoe in 1944.

The results of conservative surgical treatment of acquired hæmatometra are usually good. The menstrual function returns to normal, but subsequent pregnancy is not common.

Prevention of the condition can be summarized in the statement of Melody in reference to the treatment of cervical stenosis—"the avoidance of trauma to the cervix in all obstetrical and gynæcological practice".

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STATUS EPILEPTICUS OCCURRING DURING CORTISONE THERAPY.

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PRACTITIONERS are clamouring at the present time to obtain, from the limited quantity available, supplies of the hormone substances for the treatment of divers disorders. We report the following case in order to sound a note of warning that these "wonder drugs" are not without their risks and that their use is not without possible complications.

Clinical Record.

G.E., a girl, aged fifteen years, was brought to Sydney under the Far West Children's Health Scheme for treatment of her arthritis. She had been in a country hospital for six months. Her previous history gave no suggestion of convulsive seizures, epileptic variants or head injury.

On her admission to the Eastern Suburbs Hospital in August, 1950, she showed the polyarthritis, muscular wasting, anæmia, lymphadenopathy and splenomegaly characteristic of the syndrome of Still's disease. Her hæmoglobin value was estimated to be 7·3 grammes *per centum* (52%), and the erythrocyte sedimentation rate was 44 millimetres in one hour.

With physiotherapy, blood transfusions and the administration of iron, thyreoid extract and vitamins, the patient improved sufficiently to be up and about and to use her hands well. A course of "Primacort" and vitamin *C* proved of little benefit. Her condition, after this initial improvement, remained stationary. Further blood transfusions were unsuccessful in materially raising her hæmoglobin level and the sedimentation rate remained about the same as on her admission to hospital.

A course of cortisone therapy was now decided upon. At this stage the girl's weight was five stone six pounds and her blood pressure was 125 millimetres of mercury, systolic, and 75 millimetres, diastolic. She was given 50 milligrammes of "Cortone" twice a day for one day, then every six hours for five days, then twice a day for two days; then she was given 37·5 milligrammes twice a day for two days; the total dosage was 1350 milligrammes in ten days. During this period her daily blood pressure readings showed no rise, the urine remained clear and no peripheral œdema appeared.

Towards the end of her treatment some observers had the impression that the left side of the face was more rounded than the right. This had not been appreciated before. She had gained three pounds in weight since the commencement of cortisone therapy. During this period the patient felt much better and was very conscious of improvement in the affected joints. Swelling was considerably reduced and there was a great improvement in their mobility. However, the girl's demeanour remained one of quiet depression.

During the night of the tenth day of treatment she complained of nightmares and a headache, but later slept as usual. On the following morning she could not be roused, and about an hour later had a typical epileptiform convulsion. After a brief interval fits recurred until typical *status epilepticus* was produced, which required anæsthesia for its control. At this stage she was transferred to the Sydney Hospital.

After her admission to this hospital *status epilepticus* recurred and a barbiturate was given intravenously. Under this anæsthesia lumbar puncture was performed. The fluid was crystal clear and under a pressure of 110 millimetres. A smart rise occurred on jugular compression. The constituents of the fluid were normal in every respect and the Wassermann test produced a negative result.

After recovery from the anæsthetic the girl was restless, confused and irrational, but steadily improved each day, until ten days after her admission to Sydney Hospital she was fit to be returned to the Eastern Suburbs Hospital. However, a striking change in the patient's demeanour was now obvious. Whereas formerly she was quiet and rather depressed, now she was bright and loquacious. This psychic change has not been maintained, but the girl is still brighter and more animated than formerly.

While she was in the Sydney Hospital an electroencephalogram revealed no evidence of focal abnormality.

Discussion.

The biochemical studies which were carried out on the blood in this case gave the following results: (i) sodium content, 374 milligrammes per 100 millilitres of serum (normal, 325 to 350 milligrammes); (ii) calcium content, 9·8 milligrammes per 100 millilitres of serum (normal, 9 to 11 milligrammes); (iii) potassium content, 16·4 milligrammes per 100 millilitres of serum (normal, 18 to 21 milligrammes); (iv) sugar content (fasting), 96 milligrammes per 100 millilitres of blood (normal, 60 to 120 milligrammes). Thus the serum sodium level was increased, which indicates a retention of this ion, while the serum potassium content was decreased. The genesis of the cerebral disturbance may have been sodium and fluid retention with production of cerebral œdema without evidence of fluid in the peripheral tissues. In retrospect the gain of three pounds in weight may thus have been of significance, whereas at the time it was ascribed to her improvement in appetite and well-being.

On the other hand, cortisone and ACTH are said to produce changes in electroencephalographic patterns, so that the convulsive seizures in this patient may have been an exaggeration of this effect and due to direct stimulation of the cerebral cortex. A third possibility is that our patient is a potential epileptic whose convulsive tendency has been unmasked by cortisone.

The occurrence of epileptiform seizures during treatment of an asthmatic by cortisone has been reported from Boston by Lowell *et alii* (1951).

From our experience it would appear that clinical examination of the patient may not be enough to detect undesirable physiological effects of treatment with hormone substances. Salt restriction and biochemical survey during therapy may be necessary. This episode determines more than ever that these potent preparations should be used only when strictly indicated and where adequate supervision is possible.

Reference.

Lowell, F. C., Franklin, W., Beale, H. D., and Schiller, I. W. (1951), "Occurrence of Convulsive Seizures during Treatment of Asthma with Cortisone Acetate", *The New England Journal of Medicine*, Volume CCXLIV, page 49.

Reviews.

CYTOLOGY AND CELL PHYSIOLOGY.

A NEW edition of the book "Cytology and Cell Physiology" has been produced by the combined effort of thirteen scientists, each contributing a chapter or part of a chapter in his own special field.[1] There are eleven chapters in all dealing with cytological techniques by J. R. Baker, F. K. Sanders and R. Barer, physical and physiochemical studies of cells by J. F. Danielli, the monolayer technique by J. H. Schulman, the cell surface and cell physiology by J. F. Danielli, nucleus, chromosomes and genes by M. J. D. White, mitochondria and the Golgi complex by G. H. Bourne, micro-incineration and the inorganic constituents of cells by E. S. Horning, enzyme systems of cells by G. Blaschko and F. K. Sanders, pathological aspects of cytology by R. J. Ludford, histogenesis in tissue culture by Honour B. Fell and some aspects of evolutionary cytology by E. N. Willmer.

The first edition of this book was written during the early part of the Second World War under difficult conditions. The present edition written under somewhat more favourable circumstances retains all the original chapters which have been revised and in some cases rewritten; two new chapters have been added. The combined writings of so many scientific specialists gather under one cover a lot of facts about the many methods which have been used in the study of cell structure and function and about the results obtained with these methods. For those requiring a more detailed account of any aspect there is a good bibliography at the end of each chapter. The book is excellently produced.

This is not a book which can be easily read and assimilated. The physiologist and the medical practitioner are mainly interested in the functioning of the organism as a whole, and in the manner in which the different systems are coordinated to maintain the well-being of that organism. But it is ultimately the complicated series of chemical reactions that occur in the cell which determine the functioning of the body as a whole. It is evident, therefore, that in trying to elucidate the problems concerned with the life of the cell, the histologist, biochemist and physiologist must collaborate and coopt the physicist and the chemist. This book shows that such collaboration exists. For example, microscopy in the study of cytology had reached almost its final stage of development, but the phase-contrast microscope and the electron microscope have opened up new fields in cytology. This is but one example of many in this book in which the development of physics and chemistry and the collaboration of the physicist and chemist are aiding the biologist to throw new light on the structure and function of the cell.

In a foreword Professor R. A. Peters commends the book to those "who wish to think more deeply about the varied problems presented by the living cell and so to approach a better understanding of the unit upon which the phenomena of life are based".

"FAREWELL TO THE COUNTESS OF CHINCHON."

IN a splendid little book, "The Conquest of Malaria",[2] Dr. J. Jaramillo-Arango, former rector of the medical faculty of Bogota, Columbia, completely disposes of the popular legend associating the Countess of Chinchon with the introduction of cinchona bark to the Old World, and he has delivered his *coup de grâce* in a thoroughly effective and authoritative manner. Ten years ago credulity was first shaken by the researches of A. W. Haggis, of Baltimore, who pointed out the many weaknesses in the chain of events as handed down by tradition; and now Dr. Jaramillo-Arango, who is acknowledged to be a gifted medical historian as well as a distinguished South American physician, has made full use of his linguistic abilities and scientific acumen in the sifting of every conceivable source of information on the subject. Hence the book is of great historical interest and importance as a fully documented, scholarly and concise account of the known facts.

Dr. Jaramillo-Arango has divided his subject into three main parts: the history of malaria with special reference to the development of the mosquito-malaria theory; the basic facts in the history of cinchona; and, lastly, the progress achieved in the prevention and treatment of the disease. The letterpress is enlivened by the inclusion of thirty-six apt illustrations showing the earliest drawings of the cinchona tree, ancient maps of the "quina-quina" country in South America, modern photographs of these localities, and portraits of the men who have contributed to our present knowledge of the ætiology, epidemiology, prophylaxis and treatment of malaria.

It is only natural that one should experience a mild exhilaration to find that Australian contributions receive more than passing mention in the unfolding of this moving narrative: the discovery of the adult filarial parasite by Joseph Bancroft in 1876, the work of Brian Maegraith at the Liverpool School of Tropical Medicine, and of N. Hamilton Fairley, Professor of Tropical Medicine in the University of London. Dr. Jaramillo-Arango gives a full account of the investigations of the Land Headquarters Medical Research Unit carried out with volunteers at Cairns in 1945, and he goes so far as to say that the Queensland experiments are comparable in importance with those of the American medical unit which grappled so successfully with the problem of yellow fever in La Havana at the beginning of this century.

There is new and interesting information about an almost forgotten pioneer in the history of malaria, Charles Ledger, who was responsible in 1864 for the surreptitious exportation from Bolivia of thousands of *Cinchona calisaya* seeds and for their disposal to the Dutch Government as the nucleus of the large plantations of a new variety, *Cinchona ledgeriana*, in the East Indies. Early in the present century, Ledger died in comparative poverty at Goulburn, New South Wales, where he had lived for many years.

There are several reasons why this well-written book should be carefully read by every Australian doctor, and no medical library should be without it.

A YEAR BOOK OF ENDOCRINOLOGY.

THE increasing number of significant papers being published on endocrinology has prompted the publishers of the Practical Medical Series of Year Books to devote one volume for the first time exclusively to this subject—"The 1950 Year Book of Endocrinology", edited by Willard O. Thompson.[1] It covers journals received by the editor between January, 1950, and January, 1951. Each gland of internal secretion (pituitary, thyreoid, parathyreoids, adrenals, testes and ovaries) has a section of its own, and three concluding sections deal with *diabetes mellitus*, potassium metabolism and miscellaneous subjects. The section on the pituitary is particularly swollen by the large amount of important work being reported on ACTH; reference is made to its metabolic effects and to its clinical application in rheumatoid arthritis and acute rheumatic fever, in allergic and pulmonary diseases, in pathological conditions in the eye, in leuchæmia and cancer, in nervous and mental diseases, in the gastro-intestinal tract and in miscellaneous conditions. Other main subjects in this section are Cushing's syndrome, the pituitary growth hormone, hypopituitarism and the posterior pituitary lobe. The material on the thyreoid is grouped under headings of physiology, hypothyreoidism, hyperthyreoidism, cancer and miscellaneous conditions; work on radioactive iodine in experimental investigation and in clinical therapy occupies pride of place. Cortisone dominates the section on the adrenals, but much other important work, especially on the cortex, is covered. The section on the testes has subsections on physiology, hypogonadism and infertility, tumours and hormones in the treatment of cancer. That on the ovaries has subsections on hormonal

[1] "Cytology and Cell Physiology", edited by Geoffrey H. Bourne; Second Edition; 1951. Oxford: Geoffrey Cumberlege, The Clarendon Press. 9¼" × 6", pp. 540, with 33 plates and many text figures. Price: 87s. 6d.

[2] "The Conquest of Malaria", by Dr. Jaime Jaramillo-Arango: 1950. London: William Heinemann (Medical Books), Limited. 8¼" × 5¼", pp. 142, with many illustrations. Price: 21s.

[1] "The 1950 Year Book of Endocrinology (January, 1950-January, 1951)", edited by Willard O. Thompson, M.D.; 1951. Chicago: The Year Book Publishers, Incorporated. 8" × 5¼", pp. 500, with 138 illustrations. Price: $5.00.

factors involved in normal and abnormal menstruation, pregnancy and infertility, ovarian tumours and hormonal treatment of cancers of the breast. The section on *diabetes mellitus* covers physiology and biochemistry, pathology, treatment and complications; the material presented underlines the continuing development in the understanding and management of this disease. A short section on potassium metabolism brings into prominence the recent appreciation of the importance of potassium in body metabolism. The editor appears to have chosen his material with care and provides a considerable help to its appreciation by the average reader with his free but discriminating comments. The only Australian authors who appear to be included are J. Bornstein and Phyllis Trewhella, of Melbourne. In a special article the editor describes developments in endocrinology between 1940 and 1950. This volume should have a wide appeal, for all parts of medicine are affected by the far-reaching developments to which it is a guide.

A YEAR BOOK OF PHYSICAL MEDICINE AND REHABILITATION.

EVEN a casual glance at "The 1950 Year Book of Physical Medicine and Rehabilitation" reveals the enthusiasm of the editor and associate editors for the subject—an enthusiasm common to most of its exponents.[1] A more careful perusal of the material in the Year Book should convince those of lesser enthusiasm that the subject is able to contribute much to therapy in many parts of medicine and surgery; the scope and future possibilities of physical medicine and rehabilitation will be found briefly set out in the summary of an address given by the Year Book's editor, Frank H. Krusen, as chairman, to the new Section on Physical Medicine and Rehabilitation of the American Medical Association. Three introductory special articles are of interest. Krusen outlines pertinent developments in physical medicine and rehabilitation during 1950, referring to the growth of the specialty as such, as well as to its literature and technical aspects. Earl C. Elkins, an associate editor, reports and comments on an investigation into educational facilities in physical medicine and rehabilitation in the United States. George G. Deaver, the other associate editor, discusses team work in rehabilitation. The abstracted material, to which a minimum of editorial comment is added, is grouped into sections on general aspects, physiological considerations, diagnostic applications, devices and mechanical procedures, exercise and massage, heat and cold therapy, ultra-violet irradiation, hydrotherapy and spa therapy, medical electricity, microwave diathermy, ultrasonics in medicine, poliomyelitis, cerebral palsy, amputees, and orthopædic, general medical, neurological and psychiatric, geriatric and pædiatric conditions. General medical conditions considered are arthritis, respiratory diseases, chronic illness, physical fitness, peripheral vascular disease and diseases of the eye and ear. The editors have combed a wide range of literature in making their selection; although liberal in their choice of material for inclusion, they are critical in their comments where necessary—for example, in the section on ultrasonics, in which most of the material is continental in origin. Australian authors represented include I. A. N. McCallum, F. H. Shaw and W. T. Agar, H. Hoffman, Jean Macnamara and Michael Kelly, all of Melbourne, R. G. C. de Crespigny, of Adelaide, and T. W. Burgess, of Sydney. With its strongest emphasis on rehabilitation, the volume is well in accord with the spirit of modern therapy. It should be of interest not only to those specially concerned with physical medicine and rehabilitation, but to all whose work touches this field.

CHILDREN'S FEET.

"YOUR CHILDREN'S FEET", by Charles A. Pratt, is a little book written to help parents in the care of their children's feet and to enable them to detect in the early stages any faults that may develop.[2] The author states that four out of every five children have some sort of foot deformity, and he attributes this mainly to bad footwear superimposed upon slight development defects. Quite rightly it is advocated that all footwear should satisfy strict standards and that all shoes should be stamped as fulfilling these requirements. In dealing with *hallux valgus* the author states: "If everybody wore footwear with a straight inner border, correctly fitted, from the cradle to the grave, the condition would become extinct." All parents can learn many useful facts from this practical book that will aid them in caring for the feet of their children.

[1] "The 1950 Year Book of Physical Medicine and Rehabilitation (December, 1949-January, 1951)", edited by Frank H. Krusen, M.D., Earl C. Elkins, M.D., and George G. Deaver, M.D.; 1951. Chicago: The Year Book Publishers, Incorporated. 8" x 5¼", pp. 328, with 134 illustrations. Price: $5.00.

[2] "Your Children's Feet", by Charles A. Pratt; 1951. London: Watts and Company. 7¼" x 4¾", pp. 62, with fourteen illustrations. Price: 7s. 6d.

Books Received.

[The mention of a book in this column does not imply that no review will appear in a subsequent issue.]

"A Short Textbook of Midwifery", by G. F. Gibberd, M.B., M.S. (London), F.R.C.S. (England), F.R.C.O.G.; Fifth Edition; 1951. London: J. and A. Churchill, Limited. 8½" x 5½", pp. 584, with 199 illustrations. Price: 25s.

The fourth edition was published in 1947.

"The Child Unborn", by R. J. Harrison, M.A., D.Sc., M.B., B.Chir., F.L.S.; 1951. London: Routledge and Kegan Paul. Sydney: Walter Standish and Sons. 9" x 6", pp. 240, with 62 illustrations. Price: 15s.

For the "educated general reader" to describe what happens in conception, pregnancy and parturition.

"A Text-Book of Medicine", edited by E. Noble Chamberlain, M.D., M.Sc., F.R.C.P.; 1951. Bristol: John Wright and Sons, Limited. 9½" x 6½", pp. 974, with 266 illustrations, some in colour. Price: 50s.

The work of seventeen contributors, the book is stated to have a clinical approach with emphasis on the basic and traditional methods of examination.

"Enzymes and Enzyme Systems: Their State in Nature", edited by John T. Edsall; 1951. Cambridge, Massachusetts: Harvard University Press. 8½" x 5½", pp. 160, with 29 figures. Price: 31s. 6d.

The first of a series of memoirs to be issued by the Laboratory of Physical Chemistry Related to Medicine and Public Health by the Harvard University.

"Skill and Age: An Experimental Approach", by A. T. Welford and members of the Nuffield Research Unit into Problems of Ageing at the Psychological Laboratory, Cambridge, with a foreword by Professor Sir Frederic C. Bartlett, C.B.E., F.R.S.; 1951 London: Geoffrey Cumberlege, Oxford University Press. Melbourne: Oxford University Press. 9" x 6", pp. 170, with nine figures. Price: 16s.

The main purpose was to study the changes of skill in middle and old age with a view to ultimate applications in industry.

"The Surgical Clinics of North America"; 1951. Philadelphia and London: W. B. Saunders Company. Melbourne: W. Ramsay (Surgical) Proprietary, Limited. Lahey Clinic Number. 9" x 6", pp. 316, with 88 illustrations. Price: £7 5s. per clinic year in cloth binding and £6 per clinic year in paper binding.

A "Lahey Clinic Number" comprising a symposium on genito-urinary surgery and a series of papers on miscellaneous subjects. The former consists of ten papers; the latter are 22 in number and deal with divergent subjects of a practical kind.

"The Medical Clinics of North America"; 1951. Philadelphia and London: W. B. Saunders Company. Melbourne: W. Ramsay (Surgical) Proprietary, Limited. New York Number. 9" x 6", pp. 306, with 62 illustrations. Price: £7 5s. per clinic year in cloth binding and £6 per clinic year in paper binding.

A "New York Number", consisting of a symposium on obstetrics and gynæcology of nineteen articles by different authors, and an additional article on Rocky Mountain spotted fever.

"Prothrombin Deficiency", by Rosemary Biggs, M.D.; 1951. Oxford: Blackwell Scientific Publications. 9" x 5½", pp. 92, with 14 text figures. Price: 10s. 6d.

A practical book for the hæmatologist, the clinician and the laboratory worker.

The Medical Journal of Australia

SATURDAY, SEPTEMBER 8, 1951.

All articles submitted for publication in this journal should be typed with double or treble spacing. Carbon copies should not be sent. Authors are requested to avoid the use of abbreviations and not to underline either words or phrases.

References to articles and books should be carefully checked. In a reference the following information should be given without abbreviation: surname of author, initials of author, year, full title of article, name of journal without abbreviation, volume, number of first page of the article. If a reference is made to an abstract of a paper, the name of the original journal, together with that of the journal in which the abstract has appeared, should be given with full date in each instance.

Authors who are not accustomed to preparing drawings or photographic prints for reproduction are invited to seek the advice of the Editor.

THE NEXT CONGRESS.

READERS of this journal already know, or should know, that the next session of the Australasian Medical Congress (British Medical Association) is to be held at Melbourne on August 22 to 29, 1952. Since the end of the Second World War the sessions of congress have once again been established as an integral part of the life of the medical profession of this country, and as an expression of the scientific aims of the British Medical Association. The sixth session at Perth in 1948 and the seventh at Brisbane in 1950 are still fresh in the memory of those who attended them and will be remembered as having their own particular attractions. No two medical congresses are quite the same in arrangement and general effect, but the atmosphere of friendly expectation and keen discussion is invariable. The meeting at Melbourne will be a notable session and special features are already being planned. Melbourne has had no congress since the first session was held there in 1923 under the presidency of the late George Adlington Syme. The enormous Melbourne gathering of 1935 was not a session of congress, but the annual meeting of the whole British Medical Association when a large delegation travelled across the world from the Old Country to be present. The president on that occasion was the late James William Barrett and Dr. J. P. Major was the honorary general secretary. With their team of officers they showed that Melbourne has a great deal to offer to a large body of medical men and women in search of communion and inspiration. On this occasion Sir John Newman Morris, the president, and Dr. C. H. Dickson, the honorary general secretary, will with their band of officers keep up the Melbourne tradition and with the cooperation of members of the Branches will enhance the reputation of the congress.

Members of the Brisbane congress were very impressed with the way in which the plenary session was conducted—it took the form of a panel discussion on rheumatism, in which questions were put to a series of experts in the subject. It was thought by many of those present that at future sessions this type of presentation should be used on more than one occasion. Some members put forward the view that in the meetings of the individual sections too many of the discussions had an appeal to specialists and that those interested in medicine as a whole did not easily find subjects within their range of experience. Clearly this view is debatable—cogent arguments for and against it can be advanced. The Executive Committee of the eighth session for next August has adopted a plan which will be something in the nature of an experiment. The discussions will as usual extend over four days. There will be no plenary session; in its place a series of panel discussions and other set discussions will be held. These will extend over two days and two or three sessions will be held concurrently—two or three in the morning and two or three in the afternoon. There will probably be five or six panel discussions; at the other discussions set papers will be presented by selected speakers and a general discussion will be held on them. The subjects tentatively chosen cover fields likely to interest every member; at the same time they will give opportunity for persons with special experience to express their views. These panel discussions will take the place of what in past sessions have been known as combined meetings of sections. The last two days of the session will be given over to meetings of sections. The list of sections is as follows: Anæsthesia; Dermatology; History of Medicine; Medicine; Naval, Military and Air Force Medicine; Neurology and Psychiatry; Obstetrics and Gynæcology; Ophthalmology; Orthopædics and Physical Medicine; Oto-Rhino-Laryngology; Pathology, Bacteriology, Biochemistry, Experimental Medicine and Forensic Medicine; Pædiatrics; Public Health, Industrial Medicine and Tropical Medicine; Radiology and Radiotherapy; Surgery.[1] Though further details of the work at panel discussions and in the sections cannot be given at this stage, members of the Branches may take it that the programme will be worthy of the occasion.

One feature of next year's Melbourne congress remains to be explained. The Victorian Branch of the British Medical Association has what this journal described some time ago as a symbiotic existence with the Medical Society of Victoria. The Branch was established in 1880 and it was then that the symbiosis began—the Medical Society of Victoria came into being in 1852. The medical profession in Victoria has every reason to be proud of its long corporate existence. It was because 1952 is the centenary year of the Medical Society of Victoria that the Victorian Branch invited the Federal Council to assign the Eighth Session to Melbourne. Throughout the Commonwealth, medical men and women will congratulate Victoria on its past record and will share its hopes for the future. This, however, is not enough. In these days when values appear to be changing and uncertainty is felt in many fields of human endeavour, members of the medical profession have many reasons to seek cohesion. Knowledge of one another and an understanding and fusion of outlook are more necessary today than ever before. Attendance at congress is one way in which we can learn to know one another; the idea is to find grounds for agreement, not points of difference. And this holds for the whole community. The date August 22 to 29, 1952, should be reserved now by members of the Branches for a journey to Melbourne.

[1] A list of the Secretaries of Sections with their addresses was published in the issue of July 21, 1951, at page 98.

Current Comment.

DIETARY DEFICIENCY OF RIBOFLAVIN.

IN 1938 Sebrell and Butler described as characteristic of riboflavin deficiency in man angular stomatitis, a sore magenta tongue, seborrhœic dermatitis about the nose and scrotum, and vascularization of the cornea. These lesions have been observed under clinical conditions in which deficiences of several essential nutrients coexisted, response to riboflavin supplementation and failure of response to other therapy being the sole criteria for the diagnosis of riboflavin deficiency. It has become generally recognized that all cases of *B* vitamin deficiencies in man are multiple vitamin deficiencies, and lack in amino acids may be present also.

There has been no satisfactory study of pure riboflavin deficiency in man until O. W. Hills, E. Liebert, D. L. Steinberg and M. K. Horwitt studied fifteen males with a diet restricted in riboflavin but in all other respects adequate.[1] Fourteen other subjects were observed simultaneously as controls. The subjects were all schizophrenics in good physical condition and of fairly good mental stability, all inmates of a hospital. They lived for the period of the experiment as a group and so were subject to almost identical environmental circumstances.

The experimental diet contained 0·55 milligramme of riboflavin in 2200 Calories and was given for nine to seventeen months. The amount of riboflavin chosen was based on two considerations. Sebrell and Butler had had positive results when 0·5 milligramme per day was given; further, it was found difficult to devise a reasonably palatable diet for long-term consumption containing less than 0·55 milligramme per day. To ensure that there was no shortage of other vitamins, a supplement containing thiamine, nicotinamide, folic acid, ascorbic acid, vitamin *A* and vitamin *D* with dicalcium phosphate and ferrous sulphate was given. Very thorough investigations of the skin, the eyes and the nervous system were carried out. The outstanding pathological findings were those observed in the skin. Not all the subjects showed all the changes. The lesion most frequently seen was dermatitis of the scrotum. Twelve of the fifteen subjects showed this in greater or less degree. Other lesions observed were seborrhœa of the scalp, the chest and around the nose, cheilosis, angular stomatitis. In most cases the lesions were not severe. The course of all the dermatological eruptions was one of exacerbations and remissions. The response to treatment with riboflavin of all subjects was prompt and in some cases dramatic. The scrotal dermatitis is interesting in several respects. Only in recent years has it been recognized as related to riboflavin deficiency. It has long been known to be present in many cases of pellagra. No evidence of circumcorneal injection or vascularization of the cornea was seen at any time. None of the subjects had any evidence of glossitis nor any "magenta" tongue, "red" tongue or loss of normal papillæ. No changes were seen in the capillary bed, and there were no neurological abnormalities. Three of the subjects failed to display any clinical signs of riboflavin deficiency in spite of fifteen months on the depleted diet. All the skin lesions seen in these subjects on a low riboflavin intake may be found in cases in which lack of riboflavin is clearly not an ætiological factor, so that no one of them can be taken as pathognomonic of riboflavin deficiency. All one can say is that certain dermatological abnormalities have a predilection for persons with hyporiboflavinosis. Riboflavin is necessary for the normal growth, indeed for the very life of cells. Probably all the *B* vitamins are involved in enzyme systems necessary for the existence of living cells, so that when the dietary deficiency of any one of them is sufficiently great and prolonged, there must be a cessation of growth of new cells and some interference with functioning of cells.

It does not seem to have been noted by writers on vitamin deficiencies and is not by the authors of this study, that the epidermis is a very rapidly growing tissue throughout life and that cells, and of course their contents, are constantly being shed from the body. The skin then must make a greater demand on available *B* vitamin supplies than any other tissue, so that when there are deficiencies one might expect changes to occur in the skin early. This will be particularly the case in those parts of the skin exposed to local trauma, and it is in these places, around the mouth and nose and scrotum, that lesions occur in riboflavin and other vitamin *B* deficiencies. Thiamine is in a special position because it is actively destroyed in living cells and has to be replaced, and the most sensitive cells, those of the central nervous system, show the first signs of depletion.

One may conclude from this study that it is impossible to make a certain diagnosis of riboflavin deficiency, that a pure riboflavin deficiency probably never exists except under rigid experimental conditions, and that not less than 0·6 milligramme of riboflavin per day is necessary in the diet of an adult male to ensure that the skin lesions do not occur.

[1] *Archives of Internal Medicine*, May, 1951.

CONFUSING ASPECTS OF INFECTIOUS MONONUCLEOSIS.

IT is well to remember that medical science, like all branches of human knowledge, is alive and growing and therefore incomplete. It is necessary to keep one's outlook supple and to remember that over-rigid definitions are seldom true in the world of living things. The disease known as infectious mononucleosis furnishes an instance of this. D. A. Goldthwait and Johan W. Eliot, in an article entitled "Confusing Aspects of Infectious Mononucleosis", report the occurrence of a mild clinical and subclinical epidemic "most probably of infectious mononucleosis" in a detachment of men sent to the arctic region.[1] A plan had been made for the study of possible hæmatological changes associated with cold exposure, in twenty-six men aged from nineteen to thirty-five years, who were transferred during the winter of 1947-1948 to a field laboratory at Churchill, Manitoba, Canada, from the Quartermaster Climatic Research Laboratory at Lawrence, Massachusetts. The finding of abnormal mononuclear cells in many of the blood films prompted a close observation of the entire group for any symptoms of infectious mononucleosis. At some time during the winter every member of the group showed abnormal mononuclear cells similar to those seen in infectious mononucleosis, while only five developed symptoms sufficient to classify their condition as clinical cases. Of these five, only one showed heterophile agglutinins, and this individual also had jaundice with enlargement of the liver and spleen. Abnormal mononuclear cells were noted 30 to 131 days before the onset of symptoms in the five clinical cases.

The authors review some previously reported epidemics of infectious mononucleosis in which there were apparently many asymptomatic and subclinical cases. This disease has been defined by the triad of a peculiar hyperplasia of the lymphoid system, the presence of abnormal lymphocytes in the circulating blood and the appearance in the blood serum of agglutinins for sheep red cells which are absorbed by ox cells but not by guinea-pig kidney. However, as Goldthwait and Eliot point out, this clear-cut entity is surrounded by a haze of uncertainty which can be dispelled only by the discovery of a causal agent.

In regard to the diagnostic criteria on which the description of infectious mononucleosis is based, it is obvious that the first, reticulo-endothelial hyperplasia, is non-specific. Also, abnormal mononuclear cells may appear in the absence of demonstrable hyperplasia. The second criterion for diagnosis is the presence of abnormal mononuclear cells. Films from the subjects described by Goldthwait and Eliot showed cells that fell into the Downey[2] classifica-

[1] *The American Journal of the Medical Sciences*, March, 1951.

[2] Downey, H., and McKinley, C. A. (1923), "Acute Lymphadenosis Compared with Acute Lymphatic Leukemia", *Archives of Internal Medicine*, Volume XXXII, page 82.

tion of Type I and Type II with a small percentage that could be classified as Type III of Downey and a few cells resembling plasma cells. Blood films showing such cells in sufficiently large numbers are almost diagnostic of infectious mononucleosis, but similar cells have been described in some other diseases, the most clear-cut instance being infectious hepatitis, where in the experimentally induced disease the cells generally appear during the first week. Similar cells have been observed in measles and rubella. One cannot but feel that the presence of abnormal mononuclear cells may well be a non-specific response to varying stimuli. When associated with the two other criteria in the triad, their presence indicates infectious mononucleosis. When they are found alone their significance is doubtful. The third criterion, the presence of heterophile antibodies for sheep red cells, is of undisputed value when the antibodies are present. Failure to detect them, however, does not exclude infectious mononucleosis, for they appear at varying stages of the disease, in widely varying titres, and persist for varying lengths of time.

The latent period observed between the appearance of abnormal cells and of symptoms is puzzling. In the series under consideration this interval varied from 31 to 131 days, and other workers have found the same phenomenon. To Goldthwait and Eliot this suggested an agent-host relationship which was disturbed by external factors to produce clinical symptoms, similar to the relationship which has been described for *herpes simplex*. However, they could find no correlation between the appearance of symptoms and the degree of physical stress or exposure to cold. The percentage or absolute number of abnormal cells was of no use in efforts to predict which individuals would develop symptoms of disease. It is, of course, obvious that in a large population this type of widespread clinical infection might explain the sporadic clinical case. While infectious mononucleosis, even in well-defined cases, is usually benign and self-limited, severe and even fatal cases have occurred. In some instances it has been associated with rupture of an enlarged spleen. For these reasons, and also because the response of the reticulo-endothelial system to infection is of the greatest interest and importance, the enigma of this curious disease or symptom-complex requires to be solved. The thoughtful and observant work of Goldthwait and Eliot is a useful contribution to the subject.

INSTRUCTION ON INFANT CARE IN SWEDEN

VARIOUS useful methods have been adopted in the individual Australian States for aiding mothers to care for their babies and for instructing them in the principles and methods involved in infant care. Those who have concerned themselves with the development of this important work may be interested in an account by Arvid Wallgren[1] of methods used in Sweden for disseminating popular instruction on infant care. Professor Wallgren refers to the usual methods of popular instruction—through schools, Press, radio, lectures, films, exhibitions, booklets, folders and so on—but comments that these methods offer no assurance that what has been heard, read or seen in this way does not run the risk of incorrect interpretation. Individual instruction will largely overcome this and enables the information to be directed to the particular problems of the hearer; but even then it is difficult to control the quality of the advice given. Information from the young mother's own mother or grandmother is very variable in standard; a competent midwife can be very useful, in countries where the service of midwives is usual and readily available; but there will be general agreement with Wallgren's statement that the best form of private instruction is that given by the visiting nurses of the children's nurseries or public clinics, in countries where this form of medico-social activity has been widely developed and where its good quality is assured. Visiting the homes frequently, the nurses can see if the mother has thoroughly understood the advice and information given to her and can correct mistakes and failures in application; also the babies are examined regularly. This method is used in Sweden. First introduced as an experiment and on a chiefly private basis to two places, it was extended in 1938, by a decision of the Swedish Parliament, to the entire country and subsidized by the Government. In ten years it was operating throughout Sweden under a homogeneous organization. The service is readily available to all mothers and, at present, receives from 80% to 90% of the total number of infants. One difficulty in further extension of the work appears to be shortage of trained staff and of persons enrolling in child care nurses' training schools.

Another, and an older, Swedish method of providing information in puericulture, as Wallgren calls it, is by demonstration courses in infant care. These courses are given in rural centres, as well as in the cities, by specially trained women instructors to women over seventeen years of age; restriction to about 20 of the number admitted to a course permits more direct instruction. Girls under the age of seventeen years are given special courses at school. The programme includes, among other subjects, the rules which the mothers should observe for themselves and in caring for their children, for example, how to bathe and clothe the baby, how to prepare his bed, how to clean the room, how to regulate breast and bottle feeding, how to wean the child and how to care for him and nourish him during his first years. They are also given advice about the education of the child. Use is made of demonstrations and practical applications, and pamphlets and books of a practical nature are supplied. These courses were started in 1914 as an experiment on the initiative of the Professor of Pædiatrics in the University of Stockholm School of Medicine. They met with general approval, and to ensure a supply of competent instructors a School for Nurses in Infant Care was started in Stockholm in 1923. This school is now under the direction of the public health authorities. Together with standard nurses' training, the students receive a thorough education in child care, in social pædiatrics, and in the theory and practice of teaching. The period of training is more than three years. The courses were given a more solid organization and a firmer economic basis in 1916 by being incorporated into the semi-governmental Association for Social Protection. (Professor Wallgren is the president of the child welfare section of this association.) The courses are requested by, and so arranged as to gain the cooperation of, local government bodies, social welfare and other local organizations, schools and so on. Most of the courses are given in rural communities or in the smaller cities. In the larger cities courses of instruction with appropriate demonstrations are given at the maternity (ante-natal) clinics of the lying-in hospitals; they consist of sixteen hours of instruction, eight devoted to lectures and eight to practice. A third type of demonstration course in child care has been organized on a trial basis in certain provinces, in which nurses of the public health department have been asked to give courses in their respective districts. The head nurse and the inspector of the provincial public health department are responsible for the technical aspects of these courses, which have proved very successful and are being extended. Since 1916 approximately 10,000 courses have been given, with a total of over 200,000 participants; this amounts to about 10% of all the women of Sweden and 25% of the women whom it was particularly desired to instruct. The figures do not include courses given in girls' schools and in general public schools. Not the least important of the effects of these courses is the fact that the women instructed and advised share their knowledge with their friends and relatives. Thus, as Wallgren expresses it, the demonstration courses are like seeds which grow and yield a rich harvest; and there is probably no form of popular medical instruction with a higher rate of yield from seed sown as sound education of mothers in child care.

[1] *Courrier*, December, 1950.

Abstracts from Medical Literature.

PATHOLOGY.

Allergic Granulomatosis, Allergic Angiitis, and Periarteritis Nodosa.

JACOB CHURG AND LOTTE STRAUSS (*The American Journal of Pathology*, March-April, 1951) state that the occurrence of a clinical syndrome of severe asthma, fever and hypereosinophilia, together with symptoms of vascular embarrassment in various organ systems, has been established. Thirteen observed cases of this syndrome form the basis of their study. The basic anatomical changes in these thirteen cases consisted of widespread vascular lesions of the type seen in *periarteritis nodosa*, and of characteristic tissue alteration in the vessel wall and in the extravascular collagen system. This tissue alteration, common to all cases here reported, comprised necrosis of eosinophilic exudate, severe "fibrinoid" collagen change, and granulomatous proliferation of epithelioid and giant cells. This is considered a histopathological entity termed "allergic granuloma". The finding of the granulomatous lesions, both within vessel walls and in connective tissue throughout the body, suggests that this syndrome constitutes an entity apart from classical *periarteritis nodosa*. This assumption was corroborated by a review of fifteen cases of *periarteritis nodosa* without asthma, in none of which were found extravascular granulomata or granulomatous vascular changes. Of frequent occurrence in this syndrome are cutaneous and subcutaneous nodules with the typical connective tissue alteration. These are of significant value for diagnosis by biopsy. Of further possible diagnostic significance is granulomatous lymphadenitis. It is suggested that other allergic syndromes (Löffler, Zuelzer, Sikl) may represent the more benign forms of allergic granulomatosis, while angiitis is its most malignant expression.

The Pathogenesis of Fibrocystic Disease of the Pancreas.

ARCHIE H. BAGGENSTOSS, MARSCHELLE H. POWER AND JOHN H. GRINDLAY (*A.M.A. Archives of Pathology*, May, 1951) state that an active extract of secretin (S_1) has been obtained in six of seven cases of fibrocystic disease of the pancreas. The failure to obtain secretin in the first case studied has been attributed to the short length of intestine available for extraction. These results render untenable the theory that a deficiency of secretin in the small intestine is the primary defect in fibrocystic disease of the pancreas. A study of five cases of fibrocystic disease of the pancreas by serial sections of the head of the pancreas and the papilla of Vater revealed atresia of the main pancreatic duct within the papilla of Vater in one case, atresia of the main pancreatic duct at its perforation of the duodenal musculature and atresia of multiple interlobular ducts in two cases, and atresia of interlobular ducts alone in two cases. The evidence derived from the present study supports the theory that congenital atresia of the ducts occurring as an embryologic malformation is the cause of fibrocystic disease of the pancreas.

Cytology of Hyperplastic Pancreatic Islets in Addison's Disease.

D. L. HINERMAN (*A.M.A. Archives of Pathology*, May, 1951) states that hyperplasia of islets of Langerhans, previously undescribed, was a striking feature in eighteen cases of Addison's disease studied. This hyperplasia was characterized by a great increase in both number and size of islets and by very active neogenesis of islet cells. An increase in all types of islet cells was present, but α and δ cells were increased to a somewhat greater degree than β cells. The β cells possessed unusually abundant β granules, which are closely related to insulin. Results of this study emphasize the statement that the islets of Langerhans are very labile organs, which readily undergo morphological changes under a variety of conditions. The presence or absence of functioning adrenal cortex appears to be one of these varying conditions.

Statistical Control in Hæmatology.

H. O. LANCASTER (*The Journal of Hygiene*, December, 1950) has applied the methods of statistical control now widely used in bacteriology to the counting of the red and the white cells of the blood. He states that in the case of the red cells, there is a crowding effect due to the fact that the size of the red cells is not negligibly small in comparison with the size of the hæmocytometer squares. It is shown that this effect disappears when blocks of sixteen small squares are compared, and that the measure of the dispersion used, namely, χ^2, is appropriate. The author has tested a series of his own counts by the method and finds satisfactory agreement with the theoretical results. Certain medical graduates and technicians also produced satisfactory results. Other technicians, however, showed evidence of selecting sets of squares arbitrarily in order to attain the measure of agreement between parallel counts that is taught in the classroom and the text-books. The author quotes certain papers by Berkson and his co-workers which show how little connexion with reality or indeed theory many of the widely used criteria of a good count have.

Carcinoma of the Rectum.

DEXTER E. GUERNSEY, JOHN M. WAUGH AND MALCOLM B. DOCKERTY (*Surgery, Gynecology and Obstetrics*, May, 1951) have studied the five-year survival rates of 255 traced patients who had carcinoma of the lower part of the sigmoid, recto-sigmoid and rectum removed by combined abdomino-perineal resection. They state that the study has shown that the prognosis decreases the nearer the lesion is situated to the *levator ani* muscle. Although it is assumed in most investigations that the poorer prognosis of these low-lying lesions is due to the extension of the carcinoma along the lateral zone of spread over the *levator ani*, no instance of such spread was found in this study of 210 surgical specimens removed by the combined abdomino-perineal operation. It is probable that necropsy study in which all the tissue in the lateral zone of spread to the pelvic wall is available would be necessary for a complete and accurate investigation of this problem. A study of the sphincter muscles in these 210 specimens revealed no evidence of involvement of the external sphincters with cancer. However, in seventeen cases, approximately two-fifths of the specimens in which the lower border of the lesion was two centimetres or less above the levators (approximately five centimetres above the anal margin), the internal sphincter muscle was involved by the tumour. A decrease in the five-year survival rate was found in patients of this group as compared with patients suffering from lesions at a comparable level but without involvement of the internal sphincter. It would appear, therefore, that when sphincter-preserving procedures are being considered, the saving of the external sphincter is not contraindicated. However, the preservation of the internal sphincter in lesions five centimetres or less above the anal margin is inadvisable.

Malignant Giant-Cell Tumour of Bone.

E. S. J. KING (*The Australian and New Zealand Journal of Surgery*, August, 1951) describes the malignant giant-cell tumour of bone as the malignant growth which contains many foreign body giant cells (osteoclasts). He states that this tumour is at first localized to one part of a bone, radiologically shows characteristic trabeculation, and histologically shows a structure closely resembling that of the benign giant-cell tumour. The features distinguishing it from the benign giant-cell tumour are the relatively uniform stroma and the presence of mitotic figures. It is the less malignant form of this tumour, and the lack of clear segregation of it from the benign giant-cell tumour that are responsible for much of the confusion regarding the benign tumour. Difficulties of histological diagnosis are due to foreign body giant cells and a superadded pleomorphic stroma occurring in malignant tumours as the result of hæmorrhage and infection. The benign giant-cell tumour does not fulfil the criterion of a neoplasm, namely, that of progressive growth, and thus its distinction from the true neoplastic form is extremely important.

Neuroappendicopathy.

NORMAN H. ISAACSON AND BRIAN BLADES (*A.M.A. Archives of Surgery*, April, 1951) state that neuromata are not unusual in the appendix and probably are the cause for the removal of a large number of so-called "chronic appendices". Two types of neuromata are considered in this study, the commoner axial or plexiform neuroma which arises from the periglandular nerve plexuses usually after migration of argentaffin cells into this area and the rather rare ganglioneuroma which arises from Meissner's and Auerbach's plexuses. The authors state that "neuroappendicopathy" is a distant entity. It usually produces a dull ache or discomfort in the right lower quadrant, with slight tenderness at McBurney's point. Nausea with or without vomiting may be present. The condition can produce pain so severe as to cause patients to roll about, and

physical examination may reveal severe tenderness and abdominal muscle spasm. In spite of the apparent acuteness of symptoms and signs, the temperature is usually normal, and the total white and differential cell counts tend to remain at normal levels. Careful questioning most often reveals a history of previous attacks. Fifty-two cases of the syndrome are reviewed, and figures demonstrating the microscopic changes are shown. The authors state that before an appendix is considered normal in a patient with symptoms referable to the appendix, multiple sections stained with the Masson trichrome technique should be examined. As the diagnosis becomes commoner pathologically, ability to evaluate the clinical symptomatology of the condition will increase. Only in this way can it be hoped to gain precision in diagnosis and to differentiate similar conditions arising not only in the appendix itself but in other portions of the body.

Pulmonary Hæmosiderosis of Cardiac Origin.

ALAN C. LENDRUM (*The Journal of Pathology and Bacteriology*, October, 1950) describes the histological changes found in the lungs in hæmosiderosis of cardiac origin. He states that the hæmosiderosis which results from prolonged left ventricular failure is indistinguishable from that due to mitral stenosis. These focal accumulations of hæmosiderin are believed to be the end result of hæmorrhages from the broncho-pulmonary anastomoses in the mucosa of the terminal bronchioles. The hæmorrhages are believed to follow varicose distension of the anastomoses brought about by raised pressure in the pulmonary arteries, the exit side of the anastomoses. With mitral stenosis the increase is absolute and persistent; with remittent left ventricular failure there are periodic falls in the bronchial-arterial pressure. From the accumulation of siderophages in the group of alveoli which constitutes the focus, soluble iron reaches the adjacent stroma and there produces damage followed by reactive changes. These in turn, by lymphatic obstruction, tend to perpetuate the accumulation and so accelerate the vicious circle.

MORPHOLOGY.

Dermal Effects of Androgen.

J. B. HAMILTON AND W. MONTAGNA (*American Journal of Anatomy*, March, 1950) state that they have reported previously that in man androgens incite growth of sebaceous glands and hairs and increased vascularity of the skin. While their investigations into the more detailed morphological study of these effects in man have been delayed on account of insufficient material, they have been afforded the opportunity for investigating the effects of androgens upon the skin of hamsters. In these animals, study can be made not only on the skin in general, but also on special cutaneous areas, the pigmented costo-vertebral spots, which are male secondary characters. The spots in normal males are larger than in females or in ovariectomized females or castrate males. In females and immature animals, the spots enlarge under stimulation by male hormones, while the sebaceous glands of these spots react profoundly. The secretogogic reactions are so much greater than in the remainder of the skin as to suggest that these areas are being viewed under higher magnification. Sebaceous glands of the general area of skin also develop extensively under the influence of androgen stimulation, although their increase in volume and cytological alterations are less than in glands of the pigmented spots. Greater quantities of sebum are found in the lumina of the acini, in the ducts and upon the surface of the skin. Androgens also stimulate the growth of coarse black hairs in the costo-vertebral spot and eventually bring about coarsening of hairs of the general area of skin. The androgens produce their results with great rapidity, most of the main features being present within seventy-two hours of androgenic stimulation. The rapidity of these alterations suggests that fluctuations in body titres of androgens are accompanied quickly by profound changes in the composition and function of cells and tissues. In support of this conclusion are the facts that in man such important indices of health as the complexion of the skin and the sedimentation rate of blood can be materially influenced by, and rapidly reflect, changes in the degree of androgenic stimulation, as well as the evidence that titres of androgens do fluctuate to some extent under normal conditions and to a great degree as a result of illness.

The Median Nerve at the Wrist.

D. KENDALL (*Brain*, March, 1950) describes some unilateral and bilateral cases of median nerve palsy and concludes that compression of the median nerve between the flexor tendons posteriorly and the carpal ligament and *palmaris longus* anteriorly is a factor in its production, and is a major factor when the condition occurs in both hands in middle-aged subjects. The unilateral cases were mild and responded well to immobilization of the wrist and protection of the palm, while the bilateral palsies were severe and were treated surgically by division of the transverse carpal ligament. The result was good in seven of eight cases. The author suggests that the operation may be compared with the freeing of peripheral nerves from scar tissue, which probably interferes with the blood supply of nerves involved.

Accessory Diaphragm.

T. B. SAPPINGTON AND R. A. DANIEL (*The Journal of Thoracic Surgery*, February, 1951) describe a unique anomaly of the diaphragm, apparently due to incomplete descent of the *septum transversum*, the septum having been split into two parts by the development of the lung bud to form an accessory diaphragm.

Cerebellar Localization.

J. JANSEN (*Journal of Comparative Neurology*, December, 1950) describes the development of the fin whale and of the cerebellum of the adult bottlenose whale, as a result of which he is able to throw light on the question of topographical and functional localization within the mammalian cerebellum in general. He states that the great size of the *lobulus simplex* in whales, of which the head claims as much as one-third of the entire body length, is consistent with the electro-physiological demonstration of the projection of tactile impulses from the head to this lobule. The modest development of the ansiform lobule, in an animal of which the hind-limbs are lacking and the fore-limbs are reduced to flippers, indicates a functional relationship of this lobule with the extremities. The enormous size of the paraflocculus in Cetaceæ points to a functional relationship of trunk and tail, while the rudimentary state of development of the flocculo-nodular lobe is in harmony with the conception that this lobe is an important centre for the maintenance of equilibrium.

Prenatal Development of Human Knee Joint.

D. J. GRAY AND E. GARDNER (*American Journal of Anatomy*, March, 1950) give a full account of the development of the knee and superior tibio-fibular joints based on a study of 45 human embryos and fœtuses ranging from the sixth week of gestation to term. They state that relatively little attention has been paid to the development of joints during the later fœtal and the postnatal periods, so that a more detailed knowledge is obviously necessary for a better understanding of the physiological processes and pathological changes which take place in them. One problem on which they sought some light was whether developmental changes were to be related to genetic or to mechanical factors. In the knee joint the early appearance of menisci and cruciate ligaments before cavities are present and the resemblance of early joints to adult form lend support to the view that the factors responsible for their development are primarily genetic. Cavities are present in the synovial mesenchyme before they appear in the blastemal portion of the interzone and are fairly definitive and extensive by the ninth week. The tissue lining these cavities arises from the synovial mesenchyme and soon comes to resemble that of the adult. It is characterized by surface cells of variable arrangement and orientation, and by subjacent connective tissue containing vascular networks. The synovial tissue covers neither the articular cartilages nor the articular portions of the menisci. The latter are quite vascular and, like the cruciate ligaments, contain many collagenous fibres by term. Vascularization of the cartilaginous epiphyses begins by the twelfth week, and ossification centres appear in them by term. The individual cavities enlarge, coalesce and form a single cavity by the fourteenth week or soon thereafter. Villi first appear between the eleventh and twelfth weeks. Fat is present as isolated cells at the seventeenth week and as lobules by the eighteenth week. The general distribution of vessels and nerves to the joint resembles that of the adult by the eleventh week, and during later development branches pervade the joint to an increasing extent. A fabella, first present in a specimen of fourteen weeks' gestation, occurred inconstantly in the older specimens. The superficial prepatellar bursa appeared for the first time at the eleventh week, and the semimembranosal and anserinal bursæ at the twelfth week. The development of the superior tibio-fibular joint, although lagging somewhat behind it, is similar to that of the knee.

British Medical Association News.

SCIENTIFIC.

A MEETING of the South Australian Branch of the British Medical Association was held at the Repatriation Hospital, Springbank, on June 21, 1951, the President, DR. C. O. F. RIEGER, in the chair. The meeting took the form of a series of clinical discussions by members of the medical and surgical staff of the hospital.

Hypertension.

DR. D. A. HICKS presented a patient, aged thirty-one years, a married man with two children, whose occupation was that of assurance supervisor. He had suffered from pneumonia in 1940. A brother, who had died at the age of fifteen years, was thought to have had Bright's disease. The patient did not smoke and had taken no alcohol for six years; before that he had drunk moderately. When admitted to hospital on May 15, 1951, he stated that he knew of no attack of acute nephritis. His history was that in 1945, when feeling quite fit, he had been medically examined for discharge from the services and was found to have albuminuria. The condition was investigated at Heidelberg and at Northfield. It was found that his blood pressure was raised and the presence of albumin in the urine persistent. Renal function was apparently good. At that time he passed urine twice during the night. He first had symptoms three years before his admission to hospital; he became very tired late in the afternoon and was not coping with his job so well. About a year later he had a "check up" and his blood pressure was found to be very high. He had treatment with calcium and later with diet. Albumin was still present in his urine. In the last two months he had been having frontal headaches—dull headaches which might spread to the back of the head and were relieved by "A.P.C."; their duration was indefinite unless he took "A.P.C.", then they lasted fifteen minutes. He had vomited on two occasions, once after dietary trouble, the other after a headache, but that time the drinking water was of doubtful quality. A week before his admission to hospital, after being in bed with a cold, he had found that the right half of the right visual field had become fogged. That condition was still present. He had not noticed dyspnœa—he had been swimming until February in competition. He still had a slight cold. He had no swelling of the ankles or other symptoms.

On examination the patient was found to be a well-built young man. His heart was not enlarged. The sounds were fast, loud and clear. The pulse was of good volume. The blood pressure was 230 millimetres of mercury (systolic) and 160 millimetres (diastolic). The arterial wall was not hardened. The lungs appeared to be normal. The liver was palpable two fingers' breadth below the right costal margin. He experienced some obscure fading off of the print to the right side on attempting to read with the right eye. Examination of the optic fundi revealed grade IV retinopathy, hæmorrhages, exudate, tortuous vessels and papilloedema. The vitreous was cloudy in the right eye. Microscopic examination of the urine on May 16 revealed occasional red blood cells (approximately four per high-power field) and scattered granular casts. An electrocardiogram on May 17 revealed a "strain pattern". There was normal sinus rhythm, the *ST-T* segment was depressed in leads I and II, and the *T* wave was inverted in leads II and III and diphasic in lead I.

Commenting on the patient's condition, Dr. Hicks referred to the variable course of acute nephritis. He said that he had encountered many cases in the past year, but few were typical. Some of the patients affected had repeated acute attacks. At that stage the duration of bed rest for a patient with persistent hæmaturia must be decided; opinions varied from six weeks to an indefinite time. The Addis count might be helpful in placing the assessment of hæmaturia on an accurate scientific basis. A follow-up examination at regular intervals seemed desirable.

Dr. Hicks said that the condition of the patient under consideration was not definitely one of chronic nephritis; the pathological findings had been discovered in the course of a routine examination. The problem with the patient was what to do about his hypertension. Should it be treated *per se*, or should the treatment be directed to symptoms only? Bed rest in itself produced falls of diastolic pressure from 160 to 120 millimetres of mercury. "Vegolysin" had had no effect until given in doses of 150 milligrammes by intramuscular injection. Then the diastolic pressure had fallen to 95 millimetres of mercury on one occasion; but that level had not been maintained, and the diastolic pressure later stayed at 110 to 115 millimetres of mercury with or without administration of the drug. There was some subjective improvement while the drug was being given, but side effects were colic and diarrhœa and excessive drowsiness. There was no appreciable change in the blood urea nitrogen content, heart size, fundi *et cetera*. Dr. Hicks remarked that their results with sympathectomy had been on the whole discouraging, and the management of the patient's condition at the present stage provided a difficult problem.

DR. M. E. CHINNER said that he felt that hexamethonium bromide was not going to be a very useful drug in the treatment of hypertension, and world opinion seemed to be hardening a little towards it. In a very limited experience with the drug he had not had very much success: one patient had left the hospital having 100 milligrammes eight-hourly and feeling symptomatically better, as he had been previously worried by severe headaches, but after returning to work for a few days had suddenly died from what was apparently a coronary occlusion; another patient having similar treatment had been so concerned about visual disturbances, dryness of the mouth and giddiness that he begged to be allowed to stop the drug; in neither case had the blood pressure been significantly lowered. Two other patients, one with malignant hypertension and another with severe headaches, were under treatment; the former was receiving no benefit from the drug, the latter was better, but Dr. Chinner thought that the bed rest and effect of the bromide salt might be effecting the improvement. The results in the few cases quoted were certainly not hopeful, but Dr. Chinner felt that his experience in the use of the drug was so small that he must continue in its use for a while longer before condemning the treatment.

DR. MARK BONNIN said that the patient almost certainly suffered from chronic nephritis, and with retinopathy, such a high diastolic pressure and a raised blood urea level his expectation of life was a matter of weeks only. He said that Dr. Hicks was well aware of the possibility of phæochromocytoma in the case, but the long history of albuminuria was much more suggestive of chronic nephritis. However, cases of phæochromocytoma sometimes presented with the clinical picture of malignant hypertension. Only the previous week Mr. L. C. E. Lindon had removed an unsuspected adrenal tumour while performing Smithwick's operation on a young hypertensive woman at the Royal Adelaide Hospital. It would be worth while trying the effect of piperoxane in a rather forlorn hope of diagnosing an adrenal tumour. If there was no evidence of phæochromocytoma then the man's only chance of living more than a few weeks would be by having Smithwick's sympathectomy performed, treatment with hexamethonium having failed already. It would be a hazardous procedure, but if it was successful, the lowering of the blood pressure might postpone renal failure and would at least save the eyesight. If the patient succumbed after the operation all that would be lost would be a few weeks of rapidly progressing invalidism. However, operating in such cases might well bring the operation into disrepute, and so the case records should make it clear that it was not an operation of election but rather a last desperate attempt to give the man his only, albeit remote, chance of survival.

DR. E. F. GARTRELL warned against hypotension during the giving of "Vegolysin" and emphasized the importance of giving the injections with the patient sitting up in bed.

Disseminated Sclerosis.

Dr. Hicks's second patient was a man, aged thirty-four years, who, when interviewed in October, 1948, had stated that in 1944 he had noticed that he was unsteady on his feet. He found that he had to look at the ground to see where he was putting his feet; he could not walk in a straight line and wandered from side to side. Also he found that he could not walk downhill, but was quite able to walk uphill. If he went out at night he was never sure where he would finish up. Also in 1944 he had noticed that he could not keep his eyes steady and found difficulty in reading, as his eyes kept wandering away. In 1945 the left hand and arm had become numb; this had gradually become worse; he could not tell what he was holding in his left hand, his fingers felt like sausages and occasionally he got tingling in the fingers. His speech had altered over the past eighteen months; he found that he was hesitant and slow and tended to run one word into another. Also during the past four years he had become forgetful, and was slow in comprehending what was said to him. He had been short-tempered lately and had seen double on occasions.

On May 8, 1951, the patient was admitted to hospital as an in-patient. He recounted his history since the onset of symptoms in 1944. He said that his sight had deteriorated and had remained poor for twelve months. Then it had improved for a time, but had been deteriorating again in the last six months. In 1948 he could not stand up, because his left leg had become weak; he had no balance; he could just walk, but the leg dragged. There was no loss of feeling or sense of position, merely weakness. No recovery from this had occurred, but his condition had not deteriorated. He had developed numbness in the left side of the body, more particularly the left arm, during the last twelve months. He was finding that he did not speak so loudly as before; others had told him that he did not speak so clearly. He said that he had to think things out a long time before he could say them. He had a trembling in the arms and mouth; he could not light a cigarette easily because he could not get the match to the box, but he could feed himself and drink a glass of water if it was poured for him. He had difficulty in defæcating, and had one motion in seven days (the result of an enema). He had no trouble with micturition.

On examination he was found to be a youngish man with a euphoric appearance and outlook, with a side-to-side tremor of the head and tremor of the hands and arms of slow period. He had a foul breath and staccato-slurring speech. He had pronounced rotary nystagmus on looking in all directions and on looking straight ahead. No diplopia or evident palsy of ocular muscles was present. The visual fields were normal. Examination of the ocular fundi revealed pronounced temporal pallor of the disks (optic atrophy). The pupils were equal and reacted to light. Other cranial nerves appeared normal. Examination of the arms showed that he had lost the fine motor coordination necessary for the finger-thumb test. Power and tone were normal. He had severe intention tremor but accurate localization in the finger-nose test. Reflexes were diminished; only the left supinator jerk was normal. He had loss of tactile discrimination. The abdominal reflexes were absent in the lower quadrant, but present in the upper. The power of the abdominal muscles was good. In the legs, tone was normal and reflexes were hyperactive. There was pronounced intention tremor, but fair localization in the heel-knee test. The plantar response was upgoing (flexor) on each side. Sensation in relation to position was normal, to touch normal, to localization and discrimination impaired, to pain normal, to heat and cold normal, and to vibration absent.

Dr. Hicks said that the diagnosis of disseminated sclerosis could be made with certainty only by following the case over a period of time. When the patient under discussion had been first examined, the picture was well advanced, and the Charcot triad, rare and late, was present. The course in any one case could not be forecast, and one should guard against giving a gloomy outlook, as remission might occur for many years. In view of the pathology it was doubtful if any of the various treatments by drugs was of value. The present patient had had "Mapharsen", vitamin B_{12} and "Prostigmin", each without effect. Physiotherapy was the only treatment of real value, aiming at reeducation and muscle tone maintenance (as some of the loss was functional only). In the present case blindness was a problem because the patient's incoordination would make education in braille difficult. Dr. Hicks pointed out that the case presented an interesting variant from the classical euphoria. The patient had the euphoria, but he also had a violent side to his nature and he had in the past done bodily harm to his wife.

(To be continued.)

Out of the Past.

In this column will be published from time to time extracts, taken from medical journals, newspapers, official and historical records, diaries and so on, dealing with events connected with the early medical history of Australia.

THE SYDNEY INFIRMARY.[1]

[*Australian Medical Journal*, July 1, 1847.]

Some time ago an attempt was made to obtain from the Legislative Council a grant of money for the erection of an operating theatre in connection with this institution. The motion was, however, opposed by the members of our profession in the House and was lost. We have, on a former occasion, referred to the necessity for such an addition. If, however, the exclusive hole-and-corner system, at present acted on by the medical officers, is to be continued, a very small room will be required, since they admit no one to their operations except those especially invited by themselves—not even their own colleagues, the District Surgeons. This is the more strange since the proposition for the theatre was strongly supported by one, at least, of the medical officers, and, we believe, with the idea of ultimately establishing a medical school. We know of no institution in England of this kind, supported by public charity, where such a want of courtesy, to say the least of it, would be shown towards their brethren by the medical officers: and we can only express our surprise that such a system should, for one moment, be tolerated by the subscribers to the Sydney Infirmary. Where there is secrecy there is something to be concealed. If the institution is for the benefit of the poor, those who support it ought to have some guarantee that justice is done to the objects of their charity, and the best and only security they can have is that of open doors. If on the other hand it is to be a normal school, where the medical officers are to learn how to treat the rich by making experiments on the poor, let the fact be known, and let us have no more of the humbug of pretending that these men are the élite of the profession, elected to the office on the ground of their superior qualifications. If the ostensible reason for the preference shown to them is not a mere pretence, instead of being afraid to let their professional brethren see their practice, they ought to and would be proud and happy to afford them every facility for doing so. If they are really the "salt" of the profession their "savour" should be accessible to their contemporaries—if they are really such shining lights, for the sake of humanity they should not be hidden under a bushel—and the less informed amongst us, the "inferior grades", should have every opportunity afforded them of learning from their example. But if, on the contrary, they are no better than their neighbours—and their hole-and-corner proceedings are a tacit acknowledgement of the fact—let them descend from the elevation to which they have been raised by an undeserved preference—let them doff their borrowed plumes, cease an assumption of unfounded superiority, and frankly acknowledge themselves liable to err, and then, should they unfortunately make a mistake, they will have earned, by their modesty, a right to expect that their errors should be leniently viewed. If they will not do this—walls have eyes and ears and tongues—their mistakes will not be hidden, with all their care, and they must expect no mercy.

[1] From the original in the Mitchell Library, Sydney.

Special Correspondence.

CANADA LETTER.

By Our Special Correspondent.

It is proposed to devote this letter to a summary of the medical research crisis which is facing universities on the North American continent.

It is becoming increasingly clear that unfettered research will soon become non-existent unless some very sound thinking is done both by universities and by research foundations. Medical research is being rapidly atomized in the sense that lay bodies with great public appeal are channelling large sums of money into investigation of diseases on a basis of public sympathy, rather than upon the basic scientific exploration to be done. It is as if medical research had suddenly become a popularity contest. In addition, World War II brought about the greatest mushrooming of so-called research seen for generations. Poorly trained personnel offered themselves for what often amounted to technical investigation, devoid of cerebration, with the unfortunate result that there exist today a whole rash of pseudo-researchers, all muscling in on research funds.

There never was a time on this continent when there were so many chromium-plated research facilities, and yet some of the seasoned workers, in this unrewarding and tough field of endeavour, feel that we were never so devoid of real thinkers. For a variety of reasons, wealthy lay groups are out to force vast programmes of "research" on puzzled and embarrassed scientists.

It is against this background of change in emphasis that *The New York Times* recently conducted a survey by means of a *questionnaire* sent to the deans of medical schools in

the United States of America, and to representative medical researchers. The replies were for the most part very informative, not to say provocative, and for the first time since the war, a clear warning has been sounded as to methods of financing research. The philosophy implied in what *The Times* calls "the magic wand" theory of medical advance has certainly been held up to ridicule, since the assumption that pouring in money will produce really basic advances is utterly false. That wise and liberal spirit in medical research, Dr. Alan Gregg, Director of the Medical Sciences of the Rockefeller Foundation, has summarized it thus: "I have personally felt that if the laymen on our funds, foundations and allocations committees could learn more about the characters of good scientists and the nature of the lives they lead, the terms laid down for various projects would be a great deal more intelligently formulated."

Some of the more flamboyant public appeals by special disease groups in the community are the worst offenders when it comes to footing the complete bill. For some time it has been recognized that a university must make a charge of 8% for overhead such as space rental, services, administrative costs *et cetera*, but it is now evident that this is not sufficient to keep a medical school, housing the research, on an even financial keel. The potential drain on the resources of the already hard-pressed medical schools can be deduced from the fact that the amount spent in the United States of America on medical research has increased from a figure of $4,000,000 in 1941 to $14,000,000 in 1948.

The source of funds is important. Today, government, at whatever level, pays for one-half of all medical school research in the United States of America. The United States Public Health Service alone pays for one-quarter of the country's total research outlay. The Dean of Medicine of New York University says that: "Much of the research—perhaps two-thirds—in medical schools and research institutes is being supported by short-term grants-in-aid from specifically defined projects." Professor H. J. Muller, of Indiana University, stated: "I am strongly of the opinion that the method at present so prevalent of granting research money for special projects (as for the study of individual diseases) does restrict the range of inquiry unduly and does divert talented workers unduly from basic to popular fields."

As the methods of obtaining research grants become more and more complicated, so is the emphasis placed upon "Directors of Research", a euphemistic term for the poor intermediary who must attempt the long-term financing of a laboratory and its workers, with short-term grants. In Canada the National Research Council has attempted to overcome this difficulty through the use of "consolidated grants" to proven centres of research such as the Banting Institute in Toronto and the Montreal Neurological Institute. However, this has led to criticism from the geographically isolated or newer medical centres, which find it hard to "break in" upon the National Research Council budget. All in all, the financing of medical research needs rethinking.

Correspondence.

PRELIMINARY ANNOUNCEMENT OF A FIVE-DAY COURSE IN ARMIDALE.

SIR: "Probably half our work is wasted since our patients are so fed from the cradle, indeed, before the cradle, that they are certain contributions to a C3 nation. Even our country people share the white bread, tinned salmon, dried milk régime. Against this the efforts of the doctor resemble those of Sisyphus."

This Medical Testament extract serves in making the preliminary announcement for the information of all who will be interested that the fourteen country local associations affiliated with the New South Wales Branch of the British Medical Association will hold in Armidale from February 4 to 8 a post-graduate course of instruction and recreation. In association with the Post-Graduate Committee in Medicine in the University of Sydney, the organizing committee are extending invitations to well-known lecturers to participate in the course. The programme provides for lectures and demonstrations in as many of the branches of a general practitioner's work as it is possible to include in a course of this short duration. The lecturers invited are well known in metropolitan and country areas, not only metropolitan teachers and specialists, but also practitioners in country areas.

It is expected that many active members of the fourteen country local associations will wish to attend this course, not only because of the benefit to be gained from sound up-to-date professional teaching and pleasant daily social contact in agreeable surroundings, but also because of their active loyalty to the Federation of Country Local Associations, which continues to endeavour to advance the standard of medical practice in extra-metropolitan areas. It is also expected that many city practising doctors will wish to escape, if only for one short week, the oppressive February heat of Sydney and Brisbane and get relief in the three thousand feet altitude of the cool New England highlands, being parents of boys and girls who attend the various Armidale boarding schools, and will, as that is the week when these schools resume after the summer vacation, take the opportunity of accompanying their children back to Armidale and spend the rest of the week attending the course.

Relaxation will be provided for on the programme by games—bowls, golf and tennis—by inspection of the Hinton Art Collection and by an organized visit to Bald Blair, as well as evening functions.

Accommodation is available on application direct to the respective hotel managers at three of Armidale's hotels, namely, Tattersalls, New England and Imperial, and also at certain of the New England University College residential hostels.

The course fee determined by the committee of the Federation of Country Local Associations, after receiving the progress report of its course organizing committee, is eight guineas for the full course of five days or two guineas for a single day.

More detailed information will be made available through THE MEDICAL JOURNAL OF AUSTRALIA, through the honorary secretary of the Federation of Country Local Associations or from the undersigned on behalf of the organizing committee.

Yours, etc.,

R. J. JACKSON.

132 Faulkner Street,
Armidale,
New South Wales.

August 13, 1951.

CHILDREN AND COMMON INFECTIOUS DISEASES.

SIR: I would like to plead for a review of our attitude towards attempts to protect, by isolation, young children from the less serious infectious diseases. I refer particularly to chickenpox, mumps and German measles, and their effects on children in the kindergarten and preparatory school groups—say from three to twelve years of age.

The present practice of requiring isolation of such cases long after the mild clinical illness has subsided, and of keeping contacts away from school while they might be developing the diseases, leads to considerable waste of their time, of their parents' time and money, and severe disorganization of the school, in which often such a high proportion of a class may be away at a time that the teaching programme has to be retarded or repeated for the benefit of the absent ones.

As far as I can see no good comes of this restriction, as the children, if susceptible, sooner or later get the illnesses referred to, and then it is more serious (particularly in the case of mumps) and the loss of time from education or from work is of much more consequence. I regard it as a matter for congratulation if a child gets these illnesses over while still in kindergarten or lower forms of preparatory school, and a matter for sympathy if isolation from them at that stage has made it possible for him to contract such a condition on the eve of his wedding, or of an important examination, or of travelling, or when engaged in trying to earn a living, often in places, or under conditions, where he cannot obtain the care and nursing required. I would be in favour of removing all such restrictions to infection and keep the children sick of such conditions at home only as long as warranted by their own clinical condition, and then permit them to return to school and infect their susceptible class-mates, without hindrance, and with approval rather than hostility from the parents of those involved.

The only obligation I would place on the school is that when German measles is known to have occurred in a class or school, the mothers of susceptible contacts should be notified of the position in case one of them might be in early pregnancy and should protect herself from infection from her child should he be going to catch it. But, of course, it follows that if such an exposure programme had been in vogue when she was at school she would have nothing to fear; and in ten or fifteen years none, or very much fewer, of these illnesses would occur among adults.

Schools and parents both expect those of us who see such cases, and who act as medical advisers to schools, to be *au fait* with maximum and minimum incubation periods, periods of infectivity, and rules about isolation for these infectious diseases; and to see that they are enforced, at least as regards other people's children; and it is plain that unless a concerted opinion of the profession will agree with the views I have set out, and cooperate in getting parents to accept them, one doctor or one school alone cannot go against the present accepted traditional efforts to postpone the evil day.

I hope, sir, through your columns to obtain some endorsements of my views, or at least no opposition to them, so one could initiate such a change of the present rules without being regarded as heretical or as acting in opposition to informed opinion.

Yours, etc.,

62 Wellington Street,
St. Kilda,
Victoria.
July 25, 1951.

G. RALEIGH WEIGALL.

ETHICAL TABLETS, PRICE LIST: FOR THE MEDICAL PROFESSION ONLY.

SIR:

I'm just an "unethical" tablet,
Only an aspirin . . . that's me;
Acclaimed in the Press
To relieve all distress,
Price ninepence, large size: two and three.

Years ago, I was *persona grata*,
Acid acetyl, you know;
And *secundem art*
For the rheumatic heart
And various structures below.

But now, how the old order changeth,
I'm a pill that's been pilled, there's no doubt;
For "ethical" tabs
Sanctified in the labs,
Have shown me my place . . . which is: out.

It doesn't seem fair to a tablet
Swallowed by millions of folks,
To be left in the cold
When a-coming-on old,
Because of these "ethical" blokes.

I wish we unethical products
Knew just where the difference lies:
Have we taken the knock
Because the old doc.
Is given more "ethic" supplies?

Ethics are ethics, we take it . . .
Fairness to large or to small;
So if *we* are unfair
And the rest, "on the square",
Why bother produce *us* at all?

Yours, etc.,

Sydney,
August 4, 1951.

ARTHUR D'OMBRAIN.

Obituary.

CLARENCE READ.

DR. CLARENCE READ, whose death was recorded recently in these columns, was what has been described as one of the old school of practitioners. He practised for many years in the same place and became the trusted friend and adviser of many people. Such men have rightly been regarded as the backbone of the profession; they live to serve their patients, and in comparison everything else weighs lightly with them.

Clarence Read was born at Finsbury Square, London, in November, 1870. He qualified in medicine in 1893, becoming a member of the Royal College of Surgeons of England and a licentiate of the Royal College of Physicians of London. After a period as a hospital resident medical officer he joined the Peninsular and Oriental Steam Navigation Company, Limited, as a ship's surgeon. He was with the company for two years, on the India run, on the China run and then on the run to Australia. In 1895 he settled in Australia and worked with the late Dr. Frizell at Strathfield, New South Wales. Acting on Frizell's advice, he commenced practice on his own account at Chatswood, New South Wales, in 1897. Two years later he married and he occupied the same home till his death. In those days what is now known as the North Shore Line was very different from what it is today. Chatswood was the terminus of the North Shore railway and the country beyond was but

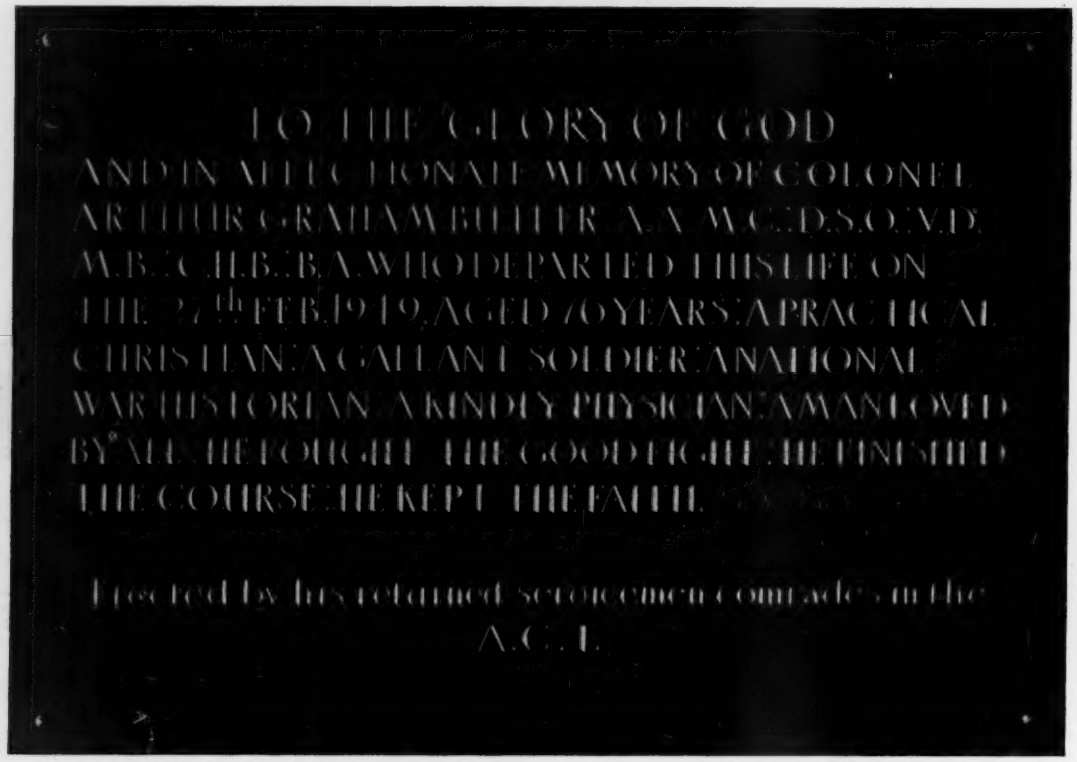
sparsely populated. Practice in Chatswood must have been like practice in a country town. Clarence Read's practice extended to Hawkesbury River in the north, covering the area between Palm Beach and St. Ives. In the other direction he joined hands with medical men from Ryde and Parramatta. Today the area forms part of the territory covered by three local medical associations, and doctors' practices may be numbered there by the score. The North Shore Hospital of those days was a small cottage hospital at Crow's Nest; it had come into being in 1885. The institution known today as the Royal North Shore Hospital of Sydney was opened with a staff of six honorary medical officers and Clarence Read was one of these. It was not until 1913 that the honorary medical staff was classified and its members allotted to special departments. Read became senior gynæcologist, and when he retired from the active staff he was appointed honorary consulting gynæcologist. When the "College of Surgeons of Australasia which Includes New Zealand", now the Royal Australasian College of Surgeons, was established in the late 1920's, Read was elected a Foundation Fellow.

Clarence Read was an active member of the British Medical Association and took a prominent part in the affairs of the New South Wales Branch. He was a member of the Council from 1906 to 1916 and from 1920 to 1921. He was president for the year 1912-1913. During his term of office the Branch was engaged in a dispute with the friendly societies. This was a disagreement of which the eventual outcome was the Common Form of Agreement, a document which was of the greatest use for many years. The dispute was the subject of a conference between government representatives, representatives of the friendly societies and representatives of the New South Wales Branch. The Honourable Frederick Flowers, the Minister, occupied the chair, and Clarence Read as president of the Branch put the case for the medical profession.

Clarence Read took an interest in the affairs of the district in which he lived, and in the process of time he became the friend and adviser of many of his colleagues who know that his example was worthy of emulation. It is men of his calibre who are needed today.

A MEMORIAL TO THE LATE ARTHUR GRAHAM BUTLER.

ON Sunday, June 3, 1951, a memorial plaque was unveiled to the memory of the late Colonel Arthur Graham Butler in Saint John the Baptist's Church, Canberra, by Air Vice-Marshal Sir Victor Hurley, K.B.E., C.B., C.M.G. The church service was arranged by the Canberra Sub-Branch of the Returned Sailors', Soldiers' and Airmen's Imperial League of Australia. The congregation included representatives of ex-service organizations, of the Australian War Memorial Board, and of the medical profession. The service was conducted by the Venerable Archdeacon R. E. Davies, and the lessons were read by Mr. H. K. Joyce, president of the Canberra Sub-Branch of the Returned Sailors', Soldiers' and Airmen's Imperial League of Australia, and by Mr. A. W. Bazley.



Sir Victor Hurley, in unveiling the plaque, a picture of which is reproduced in the accompanying illustration, said that he counted it a great privilege to have been given the honour of unveiling the memorial plaque to his old friend and comrade, Colonel A. Graham Butler. Colonel Butler had also been well known to the members of the congregation. The plaque had been erected by his returned servicemen comrades in Canberra. It was most fitting that his name and work should thus be recorded at the national capital, where he lived and worked for so many years, and in the historic church where he regularly worshipped.

Sir Victor Hurley went on to say that his friendship with Colonel Butler went back to the time when they were both medical officers in the First Australian Division which left Australia late in 1914 for the first World War. Arthur Graham Butler came of an old Queensland family, and after his preliminary schooling at Ipswich Grammar School he went to Cambridge, where he first graduated in arts, gaining first-class honours in the natural history tripos. He then went to London, where he graduated with distinction in medicine from Saint Mary's Hospital. Both at Cambridge and in London he also excelled in athletics; he rowed in the Saint John's College boat and was also an outstanding quarter-mile and half-mile runner. After his return to Australia he practised in Brisbane for many years, and his outstanding qualities were widely recognized. He was amongst the first to volunteer in the first World War, and left Australia in 1914 as medical officer to the Ninth Battalion; the battalion was recruited in Queensland and was one of the units of the famous Third Infantry Brigade which led the assault at Anzac on the morning of April 25, 1915. Sir Victor Hurley said that he believed that Colonel Butler was the first medical officer ashore, and many were the stories told of his heroism and devotion to duty during those first few critical days when the fate of the landing hung in the balance. He always showed an utter disregard for his own safety and personal comfort, and devoted himself to the well-being of the officers and men under his care. They trusted him implicitly, and knew that in him they had someone on whom they could rely in time of need. He was awarded the D.S.O. for his services at the landing and in the Gallipoli campaign which followed, and never was an honour more worthily earned.

As the war progressed Colonel Butler's quality was recognized, and he was promoted to positions of greater responsibility. He was for a time Deputy Director of Medical Services at Australian Corps Headquarters in France, and rendered outstanding service, especially during the bitter 1916 winter on the Somme and the grim fighting at Passchendaele in 1917. Towards the end of the war he was selected to collect the information and records required to produce the history of the Australian Army Medical Services in the war. Nobody then realized—nor did Butler himself—the magnitude of the task. The records were in many cases incomplete, and some of the persons who knew were no longer available to give their stories of what had actually happened. However, he proceeded to get the information at first hand by visiting the units in the field and questioning the officers and men who had taken part in the various stages of the campaigns. He moved about from unit to unit with all his belongings in an infantry pack. An office was also established in London, where the records were sorted out and collected, and in this work he had the assistance of Mr. Withers and Mr. Treloar, who were later associated with him at the Australian War Memorial at Canberra.

After his return to Australia Butler set about the monumental task of writing his history. The work was expected to take a few years at most; but it was to absorb the greater part of his working life for the next twenty years. For part of that time he was medical officer to the Royal Military College at Duntroon. He wrote and rewrote the history, because he was determined that it should be completely accurate, and he himself checked and rechecked it. The work involved him in considerable financial sacrifice, as the undertakings he had accepted at the commencement of the work proved entirely inadequate for the years of work to be done; but he never complained. Sir Victor Hurley said that the work in three volumes was regarded in military medicine as a classic, and its value was well appreciated by all seeking authoritative information. The Federal Council of the British Medical Association in Australia recognized Colonel Butler's outstanding services by awarding him its gold medal in 1944—the highest honour it could bestow, and one very rarely given.

In his later years Colonel Butler fell on ill health, and his eyesight failed owing to double cataract, so that he could read only with the aid of a large hand glass. However, he continued his work untiringly and also interested himself in all matters affecting the welfare of ex-servicemen. He was an active member and office-bearer of the Canberra Sub-Branch of the Returned Sailors', Soldiers' and Airmen's Imperial League of Australia, which he represented at various conferences of ex-servicemen.

That was the man to whose memory they did honour. His was a unique personality, in which sincerity, courage, devotion to duty and love of his fellow men were combined to a rare degree. Everyone who worked with him appreciated those qualities; he was incapable of any ungenerous or unchristian act, but he stood firmly by his principles and would roundly condemn any deviation from the path of duty and honourable conduct. His own high standard of personal conduct, so modestly and unobtrusively displayed, stimulated others to follow his example. Generous to a fault in recognizing and acclaiming merit in others, he would modestly disdain credit for his own work. The inscription on the plaque worthily expressed what they all felt:

> To the Glory of God and in affectionate memory of Colonel Arthur Graham Butler, A.A.M.C., D.S.O., V.D., M.B., Ch.B., B.A.: who departed this life on 27th February, 1949, aged 76 years, a practical Christian, a gallant soldier, a national war historian, a kindly physician, a man loved by all. He fought the good fight, he finished the course, he kept the faith.

The sermon was preached by the Bishop of Canberra and Goulburn, the Right Reverend E. H. Burgmann, D.D. In the course of his remarks, Dr. Burgmann said that he had first become acquainted with Colonel Butler during the financial depression. During this trying period Butler had had his soul torn with the tragedy of the whole situation. The thing that amazed Dr. Burgmann was the way in which he translated impulses of his generous soul into immediate action. He had a direct Christian soul; he was concerned about bringing discussions down to earth in the most practical way possible. He wanted peace on earth and goodwill among men with all his great, strong and simple heart. He realized that peace could not be secured by crying peace. He proclaimed strongly that the condition to bring peace was the condition of righteousness in human relations—that trust had to be established in the roots of all national life.

LESLIE HAROLD FOY.

WE regret to announce the death of Dr. Leslie Harold Foy, which occurred on August 25, 1951, at Corrimal, New South Wales

Notice.

THE ARCHIBALD WATSON MEMORIAL LECTURE.

THE Honorary Secretary of the New South Wales State Committee of the Royal Australasian College of Surgeons wishes to announce that the third Archibald Watson Memorial Lecture will be delivered by Dr. W. E. L. H.

DISEASES NOTIFIED IN EACH STATE AND TERRITORY OF AUSTRALIA FOR THE WEEK ENDED AUGUST 11, 1951.[1]

Disease.	New South Wales.	Victoria.	Queensland.	South Australia.	Western Australia.	Tasmania.	Northern Territory.[2]	Australian Capital Territory.	Australia.[3]
Ankylostomiasis	*	..	..	..	..	..	..	..	..
Anthrax	*	..	..	..	..	..	..	..	..
Beriberi	*	*	*	*	*	*	..	*	..
Bilharziasis	*	..	..	..	..	..	..	..	..
Cerebro-spinal Meningitis	1(1)	4(1)	1(1)	..	..	..	..	..	6
Cholera	..	..	..	..	..	..	..	..	..
Coastal Fever(a)	*	*	..	*	*	*	*	*	..
Dengue	*	*	*	*	*	*	..	*	..
Diarrhœa (Infantile)	..	..	3(2)	..	..	..	..	..	3
Diphtheria	2(2)	4(1)	2	6(6)	7(6)	..	..	..	21
Dysentery (Amœbic)	*	..	..	..	..	..	..	..	..
Dysentery (Bacillary)	*	..	..	..	..	..	..	..	..
Encephalitis Lethargica	..	..	..	..	..	..	..	..	..
Erysipelas	*	*	*	..	*	*	..	..	..
Filariasis	*	..	..	*	*	*	*	*	..
Helminthiasis	*	..	*	*	..	*	*	*	..
Hydatid	*	..	*	*	..	*	*	..	..
Influenza	*	*	*	..	*	*	..	*	..
Lead Poisoning	*	*	..	*	*	*	*	*	..
Leprosy	..	..	..	..	4	..	..	..	4
Malaria(b)	*	..	2(2)	..	..	..	..	..	2
Measles	*	*	*	103(48)	*	*	..	..	103
Plague	..	..	..	..	..	..	..	..	..
Poliomyelitis	12(5)	14(6)	17(9)	55(23)	4(4)	1	..	..	103
Psittacosis	*	..	*	..	*	*	*	*	..
Puerperal Fever	..	..	..	..	..	..	..	..	..
Rubella(c)	*	1(1)	..	*	2	*	..	..	3
Scarlet Fever	23(15)	26(15)	1(1)	15(6)	2(2)	7(3)	..	..	74
Smallpox	..	..	..	..	..	..	..	..	..
Tetanus	*	..	1	*	*	*	*	*	1
Trachoma	*	..	*	*	*	*	*	*	..
Tuberculosis(d)	35(29)	30(22)	17(7)	5(3)	6(6)	2(1)	..	..	95
Typhoid Fever(e)	..	..	..	..	1(1)	1(1)	..	..	2
Typhus (Endemic)(f)	..	..	1	..	..	..	..	..	1
Undulant Fever	..	..	1(1)	..	..	..	..	..	1
Weil's Disease(g)	*	*	2	*	*	*	*	*	2
Whooping Cough	*	*	*	..	*	*	..	..	..
Yellow Fever	..	..	..	..	..	..	..	..	..

[1] The form of this table is taken from the *Official Year Book of the Commonwealth of Australia*, Number 37, 1946–1947. Figures in parentheses are those for the metropolitan area.
[2] Figures not available.
[3] Figures incomplete owing to absence of returns from the Northern Territory.
* Not notifiable.
(a) Includes Mossman and Sarina fevers. (b) Mainly relapses among servicemen infected overseas. (c) Notifiable disease in Queensland in females aged over fourteen years. (d) Includes all forms. (e) Includes enteric fever, paratyphoid fevers and other Salmonella infections. (f) Includes scrub, murine and tick typhus. (g) Includes leptospiroses, Weil's and para-Weil's disease.

Crowther at the Stawell Hall, 145 Macquarie Street, Sydney, on Tuesday, September 11, 1951, and not on Wednesday, September 12, as announced elsewhere.

UNCLAIMED MEDICAL EQUIPMENT.

ON June 20, 1951, some medical equipment was found in a flour bag at the side of Lake Road, Middle Park, Victoria. The equipment found was as follows: one case containing an electric proctoscope with battery, one stethoscope, a blood pressure apparatus, a head light transformer, a diagnostic set. So far the Victorian police have been unable to find the owner. The owner of these articles should apply for them to the officer-in-charge, Middle Park Police Station, Victoria.

LECTURE BY SIR EDWARD MELLANBY.

THE University of Sydney announces that a lecture will be given at the Wallace Theatre, University of Sydney, on Wednesday, September 19, 1951, at 9 o'clock a.m., by Sir Edward Mellanby, formerly secretary of the Medical Research Council of Great Britain. The title of the lecture will be "The Future of Medical Science". Medical practitioners are invited to be present.

Post-Graduate Work.

THE POST-GRADUATE COMMITTEE IN MEDCINE IN THE UNIVERSITY OF SYDNEY.

Films.

THE Post-Graduate Committee in Medicine wishes to announce that the following films have been added to its library on loan on behalf of the British Council:

"Techniques in Plastic Surgery (Parts I, II, III, IV, V and VI)."

"Technique of Artificial Pneumothorax."

Applications for the loan of these films should be made to the Post-Graduate Committee in Medicine, 131 Macquarie Street, Sydney. Telephone: BU 5238, BW 7483.

Week-End Course at Albury.

The Post-Graduate Committee in Medicine, in conjunction with the Border Medical Association, will hold a week-end course at the Albury District Hospital, Albury, on Saturday and Sunday, September 15 and 16, 1951. The programme is as follows:

Saturday, September 15: 2 p.m., registration; 2.30 p.m., "Scope and Possibilities of Cortisone Therapy", Dr. T. M. Greenaway; 4 p.m., "Pancreatitis (Acute and Chronic): Its Ætiology, Diagnosis and Treatment", Dr. Stanley Lovell.

Sunday, September 16: 10 a.m., "Spinal Compression", Dr. Eric Susman; 11.30 a.m., "Skin Changes in General Medicine", Dr. T. M. Greenaway; 2.30 p.m., "Resuscitation of the Shocked Patient", Dr. Stanley Lovell; 4 p.m., "Hypertension", Dr. Eric Susman; 5 p.m., question period.

The fee for the course will be £2 2s. Those wishing to attend are requested to notify Dr. F. G. Favaloro, Honorary Secretary, Border Medical Association, 543 Kiewa Street, Albury, as soon as possible.

Nominations and Elections.

THE undermentioned has applied for election as a member of the New South Wales Branch of the British Medical Association:

Leeser, Felix, registered in accordance with the provisions of Section 17 (*b*) of the *Medical Practitioners Act*, 1938-1945, 243 Elizabeth Street, Sydney.

Medical Appointments.

Dr. F. W. Williams has been appointed public vaccinator for the Shire of Phillip Island, Victoria.

Dr. J. R. Magarey has been appointed honorary visiting medical officer (surgical) to the Northfield wards of the Royal Adelaide Hospital.

Dr. H. C. Murphy has been appointed visiting medical officer to the Diamantina Receiving Depot and the Infants' Home, Wooloowin, Queensland.

Diary for the Month.

SEPT. 10.—Federal Council of the B.M.A. in Australia—Sydney.
SEPT. 14.—Queensland Branch, B.M.A.: Council Meeting.
SEPT. 18.—New South Wales Branch, B.M.A.: Medical Politics Committee
SEPT. 19.—Western Australian Branch, B.M.A.: General Meeting.
SEPT. 20.—New South Wales Branch, B.M.A.: Clinical Meeting.
SEPT. 20.—Victorian Branch, B.M.A.: Executive Meeting.
SEPT. 25.—New South Wales Branch, B.M.A.: Ethics Committee.

Medical Appointments: Important Notice.

MEDICAL PRACTITIONERS are requested not to apply for any appointment mentioned below without having first communicated with the Honorary Secretary of the Branch concerned, or with the Medical Secretary of the British Medical Association, Tavistock Square, London, W.C.1.

New South Wales Branch (Medical Secretary, 135 Macquarie Street, Sydney): All contract practice appointments in New South Wales.

Victorian Branch (Honorary Secretary, Medical Society Hall, East Melbourne): Associated Medical Services Limited; all Institutes or Medical Dispensaries; Australian Prudential Association, Proprietary, Limited; Federal Mutual Medical Benefit Society; Mutual National Provident Club; National Provident Association; Hospital or other appointments outside Victoria.

Queensland Branch (Honorary Secretary, B.M.A. House, 225 Wickham Terrace, Brisbane, B17): Brisbane Associated Friendly Societies' Medical Institute; Bundaberg Medical Institute. Members accepting LODGE appointments and those desiring to accept appointments to any COUNTRY HOSPITAL or position outside Australia are advised, in their own interests, to submit a copy of their Agreement to the Council before signing.

South Australian Branch (Honorary Secretary, 178 North Terrace, Adelaide): All Lodge appointments in South Australia; all Contract Practice appointments in South Australia.

Western Australian Branch (Honorary Secretary, 205 Saint George's Terrace, Perth): Norseman Hospital; all Contract Practice appointments in Western Australia. All government appointments with the exception of those of the Department of Public Health.

Editorial Notices.

MANUSCRIPTS forwarded to the office of this journal cannot under any circumstances be returned. Original articles forwarded for publication are understood to be offered to THE MEDICAL JOURNAL OF AUSTRALIA alone, unless the contrary be stated.

All communications should be addressed to the Editor, THE MEDICAL JOURNAL OF AUSTRALIA, The Printing House, Seamer Street, Glebe, New South Wales. (Telephones: MW 2651-2.)

Members and subscribers are requested to notify the Manager, THE MEDICAL JOURNAL OF AUSTRALIA, Seamer Street, Glebe, New South Wales, without delay, of any irregularity in the delivery of this journal. The management cannot accept any responsibility or recognize any claim arising out of non-receipt of journals unless such notification is received within one month.

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